THE IMPACT OF PHARMACEUTICAL CARE SERVICES ON THE MANAGEMENT OF ASTHMA PATIENTS IN A PRIMARY HEALTH CARE CLINIC

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THE IMPACT OF PHARMACEUTICAL CARE SERVICES ON THE MANAGEMENT OF ASTHMA PATIENTS IN A PRIMARY HEALTH CARE CLINIC

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Submitted in fulfillment of the requirements for the degree

MAGISTER PHARMACIAE

in the

FACULTY OF HEALTH SCIENCES

at the

NELSON MANDELA METROPOLITAN UNIVERSITY, PORT ELIZABETH, SOUTH AFRICA

January 2007

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Mrs B. Gold
ACKNOWLEDGEMENTS

I would like to thank the following individuals for their cooperation, dedication and talents. Without you, a project like this would never have been completed.

To the following people I am forever grateful:

- Mrs SF Burton and Mrs B Gold, my supervisors, for their continuous guidance and enthusiasm;

- Staff and patients at the Kruisfontein Clinic, for their cooperation and participation;

- My parents, my greatest teachers, and in particular my mom whose assistance was invaluable in completing this project;

- Mr David Friskin for his statistical expertise;

- Reverend Ron Burton for modifying the plastic bottles with great effort;

- My friends, for their patience, support and encouragement

I would also like to thank the Nelson Mandela Metropolitan University and the Foundation for Pharmaceutical Education for the financial assistance I received.
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# ABBREVIATIONS

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>AGKQA</td>
<td>Asthma General Knowledge Questionnaire for Adults</td>
</tr>
<tr>
<td>ALLSA</td>
<td>Allergy Society of South Africa</td>
</tr>
<tr>
<td>ANCOVA</td>
<td>Analysis of Covariance</td>
</tr>
<tr>
<td>ANOVA</td>
<td>Analysis of Variance</td>
</tr>
<tr>
<td>ATP</td>
<td>Adenosine Triphosphate</td>
</tr>
<tr>
<td>AQLQ</td>
<td>Asthma Quality of Life Questionnaire</td>
</tr>
<tr>
<td>AQLQ(S)</td>
<td>Asthma Quality of Life Questionnaire Short Form</td>
</tr>
<tr>
<td>BHR</td>
<td>Bronchial Hyperresponsiveness</td>
</tr>
<tr>
<td>BTPS</td>
<td>Body Temperature, Ambient Pressure, and Saturated with Water Vapour</td>
</tr>
<tr>
<td>cAMP</td>
<td>Adenosine 3’5’-cyclic monophosphate</td>
</tr>
<tr>
<td>CFC</td>
<td>Chlorofluorocarbon</td>
</tr>
<tr>
<td>COPD</td>
<td>Chronic Obstructive Pulmonary Disease</td>
</tr>
<tr>
<td>DPI</td>
<td>Dry-Powder Inhaler</td>
</tr>
<tr>
<td>EC</td>
<td>Eastern Cape</td>
</tr>
<tr>
<td>EDL</td>
<td>Essential Drugs List</td>
</tr>
<tr>
<td>FEF₂₅₋₇₅</td>
<td>Forced Expiratory Flow during 25-75% FVC</td>
</tr>
<tr>
<td>FEV₁</td>
<td>Forced Expiratory Volume in one second</td>
</tr>
<tr>
<td>FVC</td>
<td>Forced Vital Capacity</td>
</tr>
<tr>
<td>FS</td>
<td>Free State</td>
</tr>
<tr>
<td>GINA</td>
<td>Global Initiative for Asthma</td>
</tr>
<tr>
<td>GM-CSF</td>
<td>Granulocyte-Macrophage Colony Stimulating Factor</td>
</tr>
<tr>
<td>GP</td>
<td>Gauteng</td>
</tr>
<tr>
<td>HFA</td>
<td>Hydrofluoralkane</td>
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<tr>
<td>Abbreviation</td>
<td>Full Form</td>
</tr>
<tr>
<td>--------------</td>
<td>-----------</td>
</tr>
<tr>
<td>ICS</td>
<td>Inhaled Corticosteroids</td>
</tr>
<tr>
<td>IgE</td>
<td>Immunoglobulin Class E</td>
</tr>
<tr>
<td>IL-1</td>
<td>Interleukin-1</td>
</tr>
<tr>
<td>KASE-AQ</td>
<td>Knowledge Attitude and Self-efficacy Asthma Questionnaire</td>
</tr>
<tr>
<td>KZN</td>
<td>KwaZulu-Natal</td>
</tr>
<tr>
<td>LLN</td>
<td>Lower Limit of Normal</td>
</tr>
<tr>
<td>LTD$_4$</td>
<td>Leukotriene D$_4$</td>
</tr>
<tr>
<td>LP</td>
<td>Limpopo</td>
</tr>
<tr>
<td>LSA</td>
<td>Local Services Authority</td>
</tr>
<tr>
<td>MDI</td>
<td>Metered Dose Inhaler</td>
</tr>
<tr>
<td>MID</td>
<td>Minimal Important Difference</td>
</tr>
<tr>
<td>MMAD</td>
<td>Mass Median Aerodynamic Diameter</td>
</tr>
<tr>
<td>MP</td>
<td>Mpumalanga</td>
</tr>
<tr>
<td>mRNA</td>
<td>Messenger Ribonucleic Acid</td>
</tr>
<tr>
<td>NAEPP</td>
<td>National Asthma Education and Prevention Plan</td>
</tr>
<tr>
<td>NC</td>
<td>Northern Cape</td>
</tr>
<tr>
<td>NMMU</td>
<td>Nelson Mandela Metropolitan University</td>
</tr>
<tr>
<td>NSAID</td>
<td>Non-Steroidal Anti-inflammatory Drugs</td>
</tr>
<tr>
<td>NW</td>
<td>North West</td>
</tr>
<tr>
<td>PDE</td>
<td>Phosphodiesterase</td>
</tr>
<tr>
<td>PEFR</td>
<td>Peak expiratory flow rate</td>
</tr>
<tr>
<td>PHC</td>
<td>Primary Health Care</td>
</tr>
<tr>
<td>PTB</td>
<td>Pulmonary Tuberculosis</td>
</tr>
<tr>
<td>QOL</td>
<td>Quality of Life</td>
</tr>
<tr>
<td>SR</td>
<td>Slow Release</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Description</td>
</tr>
<tr>
<td>--------------</td>
<td>------------------------------</td>
</tr>
<tr>
<td>SRS-A</td>
<td>Slow-Reacting Substance of Anaphylaxis</td>
</tr>
<tr>
<td>TB</td>
<td>Tuberculosis</td>
</tr>
<tr>
<td>WC</td>
<td>Western Cape</td>
</tr>
<tr>
<td>ZA</td>
<td>South Africa</td>
</tr>
</tbody>
</table>
ABSTRACT

Optimal management of a chronic disease, like asthma, requires the active participation of patients. To achieve this, patients require education about asthma. Many of the recommended components of asthma care and management might not be effective without adequate patient education. Pharmacists in community, hospital and clinic practice are well placed to provide continued information and reinforcement of key messages, in order to improve compliance with medication and the outcomes of asthma management plans. Pharmacists may be able to increase medication adherence with patient counselling and monitoring systems and by facilitating communication with physicians.

However, regardless of this, it remains uncertain whether pharmacist-patient interactions improve patient outcomes, and in spite of recommendations for teamwork and a multidisciplinary approach in the education of asthma patients, medical doctors and nurses are still largely responsible for carrying out the greatest part of patient education.

The objectives of this study were therefore to determine the impact of pharmaceutical care services at a primary health care level on the management and well-being of asthmatic patients; to determine the effect of complex or multi-faceted pharmaceutical interventions, in patients with asthma, on lung function, asthma knowledge, attitudes and perceived self-management efficacy, asthma related quality of life and asthma control; and to determine the extent to which pharmacotherapeutic interventions, with regards to medication changes and dosage changes, are accepted and implemented by doctors.

A randomised-control study was conducted at a primary health care clinic in the Eastern Cape. A total of 120 patients were allocated to two groups of sixty patients each (a Control Group and an Intervention Group). Baseline values were measured and follow-up interviews and post-intervention data collection were conducted three months afterwards for each group. Patients in the Control Group were attended to by the clinic staff as usual. Patients in the Intervention Group were educated on their
disease by a pharmacist. The use of a customised 500ml plastic bottle as a spacer was suggested and each patient’s medication was evaluated against the Standard Treatment Guidelines for the management of asthma in adults at the primary health care level and where necessary, prescribing recommendations were made.

Following assessment of the medication regimens of the patients in the Intervention Group, a total of 49 prescribing recommendations were made, of which 73% were accepted by both the doctor and patient. After educating the patients in the Intervention Group on inhaler technique, a significant improvement in technique was observed at the 3-month follow-up assessment (p<0.05).

Using a short form of the Asthma Quality of Life Questionnaire (AQLQ(S)), a significant improvement post-intervention in mean total quality of life score (p<0.05) and mean average quality of life score (p<0.05) in the Intervention Group, were demonstrated. An improvement in mean activity limitation score in the Intervention Group post-intervention was also recorded for the activity limitation subscale of the AQLQ(S) (p<0.05). On measuring changes in asthma related knowledge, attitudes and self-efficacy, using a questionnaire (KASE-AQ), a significant improvement in mean knowledge score in the Intervention Group after the intervention (p<0.05) was also shown. With regards to lung function, both vital capacity (%FVC) and expiratory flow volumes (%FEV₁) improved significantly in the Intervention Group (p<0.05).

This study therefore demonstrated that multi-faceted pharmacist interventions, including medication assessment, asthma education, education on inhaler technique and the provision of medication aids in the form of spacers, can significantly improve the management of asthma patients and improve their well-being and quality of life.

Key words: Asthma, primary health care, pharmaceutical care, pharmacist intervention, asthma education
CHAPTER 1
INTRODUCTION

The incidence of asthma has been estimated to have approximately doubled over the last ten years, affecting 3% to 5% of the adult population. Asthma is a chronic inflammatory condition of the airways in which many cells and cellular elements play a role. It is usually allergic in origin and is characterised by airways that constrict easily in response to a wide range of stimuli. In susceptible individuals, this inflammation causes recurrent episodes of wheezing, tightness of the chest, coughing and dyspnoea that are often worse at night or in the early hours of the morning.

The knowledge that inflammation plays a primary role in the pathogenesis of asthma has led to the conviction that the focus of therapy is on the prevention and suppression of the underlying inflammation. Thus current therapeutic options in asthma include the use of reliever medications (relievers) used for acute exacerbations and long-term control medications (preventers) used for the prevention of symptoms and the suppression of inflammation. Proper control of a chronic condition like asthma also requires adequate patient education. Pharmacists have extensive knowledge about drug therapy that can assist both health care providers and patients in managing and controlling asthma.

The scope of pharmacy practice has developed from a primarily dispensing role to that of including pharmaceutical care. Pharmaceutical care involves the pharmacist participating in educating the patient on their disease as well as evaluating drug therapy and ensuring patient compliance with medication regimens. The most important objective of pharmaceutical care in asthma management is improving patients’ everyday functioning, which would include their emotional as well as social well-being. A measurable improvement in a patient’s asthma-related quality of life could therefore be considered as evidence that the aim of pharmaceutical care provision in asthma has been achieved.

Several studies have found that pharmaceutical services can improve the management and control of an asthmatic patient. Outcomes of these studies include improved
satisfaction with pharmaceutical care, reduced drug costs, minimisation of drug interactions, a decrease in hospitalisations, as well as improved patient knowledge and understanding of medical terminology. Other improvements documented include improved lung function measurements, improved patient quality of life and improvements in medication compliance. However, Weinberger et al (2002) reported higher peak flow rates following a pharmacist care programme but no difference compared to monitoring alone. No differences in adherence to medication or health-related quality of life were also reported.

During exposure to the environment and circumstances at the Kruisfontein Clinic, a primary health care clinic controlled by the Kouga Local Services Authority (LSA), during undergraduate practical training, it appeared that patients attending the clinic had a high incidence of asthma. It also became evident that there was insufficient implementation of the Standard Treatment Guidelines for the management of asthma in adults at the primary health care level. Both theophylline and chronic systemic corticosteroids were used frequently to manage a patient’s asthma which is not in accordance with the Standard Treatment Guidelines. During personal communication with and counselling of patients, the poor level of asthma-related knowledge amongst patients was also noted. Patients did not know how to use a metered-dose inhaler correctly and this could have led to many patients having uncontrolled asthma, despite the use of prescribed inhalers. Correct inhaler technique is crucial in ensuring that a therapeutic amount of drug reaches the lungs. These observations were confirmed by pharmaceutical personnel of the Kouga LSA.

The primary objective of this study was therefore to determine the impact of pharmaceutical care services on the management of asthma patients in a primary health care (PHC) clinic, in Humansdorp, Eastern Cape, South Africa. Secondary objectives included determining the effect of complex or multi-faceted pharmaceutical interventions in patients with asthma, on lung function, asthma-related knowledge, attitudes and perceived self-efficacy, asthma-related quality of life and asthma control. The extent to which pharmacotherapeutic interventions, with regards to medication changes and dosage changes, are accepted and implemented by doctors were also to be determined.
A review of the relevant, background literature was undertaken and is outlined in Chapter 2. This includes an introduction to asthma, the symptoms, causes and prevalence of asthma, the measurement of asthma, including lung function measurements and questionnaires used to assess asthma status. The assessment and classification of asthma in South Africa together with the treatment and management of asthma are also outlined. Consideration is also given to the different types of inhalers used in asthma and patient education regarding asthma. An overview of pharmacists’ interventions in asthma management is also provided.

The purpose of Chapter 3 is to outline the research methodology used in completion of this study. It outlines the inclusion criteria, the process of inclusion, patient data collected at the interviews, as well as the statistical methods used in the analysis of the study results.

Chapter 4 provides the results obtained from this study, and includes discussion of these results. Results are presented as baseline characteristics, prescription assessment and intervention and lastly, post-intervention assessment. Results are further categorised as quality of life questionnaire results, knowledge, attitude and self-efficacy results, lung function measurements and inhaler technique assessment.

The conclusion, limitations and recommendations for this study are presented in Chapter 5.
CHAPTER 2
LITERATURE REVIEW

2.2 ASTHMA

2.2.1 INTRODUCTION

Since antiquity asthma has been known, yet it is a disease that still defies precise definition. The word *asthma* is of Greek origin and means “panting”.

Asthma can be defined as a chronic inflammatory condition of the airways which is usually allergic in origin and is characterised by hyperresponsive airways that constrict easily in response to a wide range of stimuli. This results in the characteristic symptoms of wheeze, tightness of the chest, cough and dyspnoea that are often worse in the early hours of the morning.

It is a highly complex inflammatory disease of the airways in which many cells and cellular elements play a role, such as eosinophils, mast cells, T-lymphocytes, macrophages, dendritic cells, neutrophils and epithelial cells. These cells can influence airway function through secretion of mediators that act either directly on the airway or indirectly through neural mechanisms. Cell-derived mediators can influence airway smooth muscle tone, modulate vascular permeability, activate neurons, stimulate mucous secretion and produce characteristic structural changes in the airway.

Structural changes include basement membrane thickening, hypertrophy and hyperplasia of airway smooth muscle, increases in goblet cell number, enlargement of sub-mucous glands and remodelling of the airway connective tissue. In susceptible individuals these inflammatory changes result in the well known signs and symptoms of asthma which are usually associated with widespread but variable airflow obstruction that is often reversible.

Despite the relatively low number of asthma deaths, 80% to 90% are preventable. Most deaths from asthma occur outside the hospital and death is rare after hospitalisation.

The most common cause of death from asthma is inadequate assessment of severity of airways obstruction by the patient or doctor and inadequate therapy.

The increase in urbanisation has lead to an increase in asthma prevalence and the most common cause of death in the hospitalised patient is due to inadequate or
inappropriate therapy.\textsuperscript{14} The key to prevention of death due to asthma is therefore education of both the patient and the health care provider.

\subsection*{2.1.2 PATHOPHYSIOLOGY}

Bronchial hyperresponsiveness (BHR), airways inflammation and a variable degree of airflow obstruction (related to bronchospasm, oedema and hypersecretion) are the major characteristics of asthma. Several factors initiate, intensify or modulate the inflammatory response of the airways and produce airways abnormalities. The immune responses mediated by immunoglobulin class E (IgE) antibodies are the most significant.\textsuperscript{3} Most of the IgE in the body is bound to the IgE receptors on mast cells. When IgE on the surface of the mast cells binds antigen, various inflammatory mediators are released from the mast cell including histamine. This produces the overall effect of inflammation stimulation.\textsuperscript{15} Asthmatic patients generally have higher IgE levels.\textsuperscript{16}

All airway cells are involved and become activated in asthma. Epithelial cells become activated by IgE-dependent mechanisms, viruses, pollutants or histamines and extensive epithelial shedding occurs in asthma. This leads to heightened airways responsiveness, altered permeability of the airway mucosa, depletion of epithelial-derived relaxant factors and loss of enzymes responsible for degrading pro-inflammatory neuropeptides.\textsuperscript{3}

Eosinophils cause the release of pro-inflammatory mediators, cytotoxic mediators and cytokines. Mucosal biopsy specimens from patients with asthma contain lymphocytes, many of which express surface markers of inflammation. Alveolar macrophages engulf and digest bacteria and other foreign materials in the normal airway and they release a number of mediators which initiate and amplify inflammation in allergic asthma. High numbers of neutrophils have also been reported to be present in patients with asthma who died from sudden-onset fatal asthma. However their exact role in asthma remains unclear.\textsuperscript{3}

Leukotrienes C\textsubscript{4}, D\textsubscript{4} and E\textsubscript{4} are liberated during the inflammatory process in the lung and constitute the slow-reacting substance of anaphylaxis (SRS-A). Leukotrienes D\textsubscript{4}
and E₄ share a common receptor (the LTD₄ receptor) that produces bronchospasm, mucous secretion, microvascular permeability and airway oedema when stimulated. LTD₄ receptor antagonists can improve symptoms and lung function in chronic asthma.³

Since asthma represents a chronic inflammatory process of the airways followed by healing, this can lead to an alteration of structure known as remodelling of the airways. This may present as fibrosis and an increase in smooth muscle and mucous gland mass.³ The exudative inflammatory process and sloughing of epithelial cells into the airway lumen also impair mucociliary transport. Expectorated mucous from asthmatic patients also tends to have a high viscosity.³

It is therefore critical to manage asthma optimally, not only for the acute effects, but also to reduce long-term effects of this condition.

2.4.3 SYMPTOMS OF ASTHMA

The classical symptoms of asthma include paroxysmal breathlessness, wheeze, cough, mucous production and sleep disturbance. Airflow obstruction in asthma occurs as a consequence of airway inflammation, mucosal oedema, neural bronchoconstriction and mucous plugging. In a minority of patients with asthma, subepithelial fibrosis and airway remodelling may supervene, resulting in irreversible airflow obstruction and the development of chronic obstructive pulmonary disease (COPD).¹⁷ It is however important to distinguish between asthma and COPD since there may be some overlap between the two conditions and the treatment of the two conditions differ.
The following table outlines the major differences between asthma and COPD:

**Table 2.1 Differences between asthma and COPD according to the South African Thoracic Society and the Allergy Society of South Africa**

<table>
<thead>
<tr>
<th>Features suggesting asthma</th>
<th>Features suggesting COPD</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ young age of onset</td>
<td>➢ long history of smoking</td>
</tr>
<tr>
<td>➢ presence of atopy and / or allergic rhinitis</td>
<td>➢ usually non-atopic</td>
</tr>
<tr>
<td>➢ diurnal and / or day to day and seasonal variation in symptoms and lung function</td>
<td>➢ insidious onset of symptoms and persistent dyspnoea</td>
</tr>
<tr>
<td>➢ often normal examination and normal/near normal spirometry while in a stable state</td>
<td>➢ slow progression of symptoms</td>
</tr>
<tr>
<td>➢ marked improvement after bronchodilator and / or 2 week trial of systemic corticosteroids*</td>
<td>➢ hyperinflation and abnormal spirometry</td>
</tr>
<tr>
<td>➢ while in a stable state, progressive deterioration in lung function over time</td>
<td>➢ while in a stable state, progressive deterioration in lung function over time</td>
</tr>
<tr>
<td>➢ 12% and 200 ml improvement in FEV₁ or 15% improvement in PEFR</td>
<td>➢ poor response to bronchodilator and / or 2 week trial of systemic corticosteroids</td>
</tr>
</tbody>
</table>

Molecular and genetic technologies have added substantially to our understanding of the pathogenesis of asthma and ongoing research continues to define genetic influences of this condition. Between 30 and 70% of children with asthma will improve markedly or become symptom-free by early childhood. Chronic disease persists in about 30 to 40% of patients and generally less than 20% develop severe chronic disease.

In adults, the majority of longitudinal studies have suggested a more rapid rate of decline in lung function, primarily forced expiratory volume in one second (FEV₁), in asthmatics than in normal volunteers. However, the annual decline is less than in smokers or patients with emphysema. Subjects with less frequent attacks and normal pulmonary function on initial assessment have higher remission rates, whereas smokers have the lowest remission and highest relapse rate. The level of bronchial hyperresponsiveness (BHR) tends to predict rate of decline in FEV₁, with a greater
decline occurring with high levels of BHR. These studies also suggest that airways obstruction in asthma may not only become irreversible but may also worsen over time due to airway remodelling. The extent to which airway remodelling occurs as well as the effectiveness of therapy in altering the course of this chronic deterioration is the subject of ongoing debate and research.

2.4.4 CAUSES OF ASTHMA

The following table outlines some of the most common causes of asthma as well as some factors which can bring on an acute asthma attack.

Table 2.2 Risk factors for the development or exacerbation of asthma

<table>
<thead>
<tr>
<th>Predisposing factors</th>
<th>Causal factors</th>
<th>Contributing factors</th>
<th>Triggers for acute exacerbations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atopy</td>
<td>Indoor allergens</td>
<td>Respiratory infections</td>
<td>Allergens</td>
</tr>
<tr>
<td></td>
<td>• Domestic mites</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Animal allergens</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Cockroach allergen</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Fungi</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Outdoor allergens</td>
<td>Pollens</td>
<td>Small size at birth</td>
<td>Respiratory infections</td>
</tr>
<tr>
<td></td>
<td>Fungi</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin and other NSAIDs</td>
<td>Diet</td>
<td></td>
<td>Exercise and hyperventilation</td>
</tr>
<tr>
<td>Occupational sensitisers</td>
<td>Air pollution</td>
<td></td>
<td>Cold weather</td>
</tr>
<tr>
<td></td>
<td>• Outdoor pollutants</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Indoor pollutants</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td>Passive smoking</td>
<td></td>
<td>Sulphur dioxide</td>
</tr>
<tr>
<td></td>
<td>Active smoking</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Foods, additives, drugs</td>
</tr>
</tbody>
</table>
2.5 PREVALENCE OF ASTHMA

Studies from 2004 show an increase in asthma prevalence worldwide of 5% to 6%, with asthma affecting 8% to 10% of children and 3% to 5% of the adult population.\(^1\) Around one out of every ten people in the Western World develops asthma at some stage in their life.\(^2\)

Asthma is now the most common chronic disease in industrialised countries and its prevalence is rising throughout the world.\(^1\) The incidence of asthma has been estimated to have approximately doubled from 1995 to 2001\(^2\), with the highest incidence occurring in children, and particularly in the youngest age groups.\(^2\) Air pollution, allergen exposure, tobacco smoke and diet have all been implicated in this increase but evidence in support of these factors is conflicting.\(^1\)

The rates of death from asthma tended to decrease since 1998, perhaps due to better management.\(^2\) The more recent use of inhaled corticosteroids (ICS) has also been associated with a significant reduction in the risk of deaths from asthma.\(^2\)

There is however still a tendency to under-diagnose asthma, and consequently underestimate the true prevalence of the disease. It is also a well-recognised fact that, paradoxically, asthma is a poorly controlled disease despite the availability of highly effective medications such as inhaled corticosteroids and long-acting \(\beta_2\)-agonists.\(^2\)

Whilst asthma prevalence is not necessarily associated with poverty, the complications of asthma are. On the one hand, self-reporting of asthma is likely to reflect some degree of under-diagnosis. On the other hand, asthma rates may be inflated by confusion with emphysema and chronic bronchitis, particularly in older age groups.\(^2\) According to the South African Demographic and Health Survey of 1998\(^2\), abnormal peak flow prevalence rates were similar in men and women (4% of the total population each). Airway limitation rates were 7% for men and 9% for women older than 15 years respectively and increased with age, particularly in men. Non-urban areas had a lower prevalence rate compared with urban areas. Population group comparisons revealed the highest rates among Whites and the lowest amongst the Black population. The association of reported asthma with level of education is
complex, with a tendency for those with intermediate education to report less asthma than those with the lowest and highest educational levels. The same study found that for reported asthma, rates among women were highest in the Western Cape and lowest in the Northern Province. Amongst men, similar rates (above 4%) were reported in the Western Cape, Eastern Cape, KwaZulu-Natal and Gauteng, with lower rates in the other provinces. In the Eastern Cape 6.9% of men suffered from airway limitation compared with 8.0% of women. A total of 3.7% of men in the Eastern Cape also had abnormal peak flow rates in comparison with 4.1% of women 15 years of age and older. It is interesting to note that men with no education showed a five times higher prevalence of abnormal peak expiratory flow rate (PEFR) than men with greater than Grade 12 education. The corresponding ratio for women was fourfold. No apparent reason for this was given.

The role of tobacco smoking in respiratory disease in South Africa was confirmed in this South African survey by the finding that at all ages, men and women, who had smoked have a higher prevalence, particularly of chronic bronchitis and also of episodic airflow limitation and abnormal PEFR, than people who had never smoked before. This is quite significant if one takes into account that 42% of adult men and 11% of adult women in South Africa are smokers as well as 10% of adolescents aged 15 to 19 years. According to this survey there was a moderately strong association in males, between occupational exposure to smoke, dust, fumes or strong smells and both airflow limitation and chronic bronchitis, and a little less so for PEFR abnormality. It was noted that these prevalence ratios were generally higher than those for smoking. An increased prevalence of airflow limitation, chronic bronchitis and abnormal PEFR was also found among those who had performed underground mining work. A strong association between a history of diagnosed tuberculosis and airflow limitation, chronic bronchitis and abnormal PEFR among men and women was also found, confirming the importance and impact of the tuberculosis epidemic in South Africa. These prevalence ratios were the highest of any of the associations investigated.

Regarding asthma specifically, the findings of this survey suggested that asthma is underdiagnosed and undertreated. A potential bias of this study was that healthy working people may have been under-represented in these household surveys. It was also difficult in a survey of this nature to distinguish asthma from COPD in adults.
Nevertheless, of the prevalence of airflow limitation reported, a significant proportion (10% to 15% of the population above 44 years of age) was likely to be due to asthma.27
2.6 MEASUREMENT OF ASTHMA

2.3.2. LUNG FUNCTION MEASUREMENTS

Guidelines for the clinical management of asthma base specific treatment recommendations on the assessment of disease severity. Thus, the accuracy of such assessments is essential for proper clinical management. The consistency of asthma severity assessment in patients with difficult-to-treat disease is unknown.

A study by Miller et al (2005)\textsuperscript{28} compared asthma severity levels assessed by two different instruments, namely the National Asthma Education and Prevention Plan (NAEPP) Guidelines and the Global Initiative for Asthma (GINA) Guidelines. According to the NAEPP guidelines, the assessment of severity depends on the subjective reporting of current daytime and night time symptoms as well as on objective evaluation of lung function by spirometry or peak expiratory flow rate (PEFR) or forced expiratory volume in one second (FEV\textsubscript{1}) before the initiation of treatment. GINA guidelines consider the current clinical features of asthma (symptoms and pulmonary function) as well as medications in determining asthma severity. The NAEPP guidelines evaluate the current degree of asthma control without adjusting for medications. GINA guidelines focus on assessing the severity of underlying disease by evaluating clinical features of asthma before treatment and then adjusting the assessment of asthma severity on the basis of the patient’s response to therapy. This study by Miller et al showed striking differences in asthma severity classification. The effect of asthma on patients, often equated to asthma severity, cannot simply be assessed by using physiological measures. There is also increasing recognition that measures of the physiological and symptom deficits and amount of asthma therapy required to achieve control may be more appropriate measures of severity.\textsuperscript{28} The results of this study suggested that improvement in the classification of asthma severity is warranted. Global asthma severity assessments should consider not only patients’ physiological and symptom measures but also recent medication and health care utilization.
One of the instruments used to measure lung function is a spirometer. On using a spirometer, the patient’s age, sex, height, and ethnic group are entered into the machine to determine normal values for comparison. The patient completely fills the lungs, hears the command from the assessor to blow out for a full six seconds, and then blows out all the air into a flow sensor (transducer) in one uninterrupted breath. The test is then performed a second time to check for repeatability, which must be within 3%, to ensure that maximum effort has taken place. The device also tracks the patient’s effort by determining the time from onset of expiration to peak flow, which is normally less than 0.1 seconds. It also monitors the contour of the expiratory flow curve, checking for irregularities such as coughs and premature closure of the glottis. Values are displayed as a percentage of the value predicted, and an interpretation is also given. Because a person cannot blow out more air than is in the lungs, it is impossible to get false high results, thus eliminating false-negative tests. Spirometers require precision within 3% before displaying forced vital capacity (FVC) and FEV₁ values, so that falsely low results also are avoided or at least mitigated.²⁹

Clinical studies³⁰;³¹ have concluded that only two measurements are needed in spirometry: FVC, a test of volume, and FEV₁, a test of flow. FVC is the amount of air that can be blown out of fully inflated lungs, and FEV₁ is the rate of expiratory air flow. Normally, 70% to 75% of the FVC is blown out in the first second; thus, the FEV₁ /FVC ratio should be >0.70 or 0.75. This single ratio can be used as a highly sensitive yet non-specific screening test for patients likely to experience a rapid decline in lung function.²⁹

Figure 2.1 and Figure 2.2 indicate flow-volume (Figure 2.1 A)²⁹ and volume-time (Figure 2.1 B)²⁹ curves in a healthy individual compared with the flow-volume (Figure 2.2 A)²⁹ and volume-time (Figure 2.2 B)²⁹ curves in a patient with moderate airways obstruction. Note that expiratory time can be visualized from the volume-time curve and that peak flow can be visualized from the flow-volume curve. Thus both curves are useful.
Figure 2.1  Flow-volume (A) and volume-time (B) curves in a healthy individual. 

**Flow-volume (A)**
- Graph showing flow rate on the y-axis and exhaled volume (L[BTPS]) on the x-axis.
- FEV₁ is indicated on the graph.

**Volume-time (B)**
- Graph showing volume (L) on the y-axis and time (s) on the x-axis.
- Table below the graph showing:
  - FVC: 5.00, LLN: 3.88
  - FEV₁: 3.79, LLN: 3.12
  - FEV₁/FVC (%): 76, LLN: 71

FEV₁ = forced expiratory volume in one second; BTPS = body temperature, ambient pressure, and saturated with water vapour; FVC = forced vital capacity; LLN = lower limit of normal.
Figure 2.2 Flow-volume (A) and volume-time (B) curves in a patient with moderate airflow obstruction\textsuperscript{29}

**A**

![Flow-volume curve](image)

- \( \text{FEV}_1 \): Forced expiratory volume in one second
- BTPS: Body temperature, ambient pressure, and saturated with water vapour
- FVC: Forced vital capacity
- LLN: Lower limit of normal

**B**

![Volume-time curve](image)

<table>
<thead>
<tr>
<th></th>
<th>Result</th>
<th>LLN</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC</td>
<td>3.61</td>
<td>3.88</td>
</tr>
<tr>
<td>( \text{FEV}_1 )</td>
<td>2.05</td>
<td>3.12</td>
</tr>
<tr>
<td>( \text{FEV}_1/\text{FVC} ) (%)</td>
<td>57</td>
<td>71</td>
</tr>
</tbody>
</table>

\( \text{FEV}_1 \): forced expiratory volume in one second; BTPS = body temperature, ambient pressure, and saturated with water vapour; FVC = forced vital capacity; LLN = lower limit of normal.
The following table illustrates a guide for grading pulmonary impairment according to the two measurements needed in spirometry:

Table 2.3  Guide for grading pulmonary impairment according to percent of predicted spirometry as per The Occupational Medicine Sub-Committee (2003)\textsuperscript{32}

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Mildly impaired</th>
<th>Moderately impaired</th>
<th>Severely impaired</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV1</td>
<td>&gt;80%</td>
<td>60-79%</td>
<td>41-59%</td>
<td>&lt;40%</td>
</tr>
<tr>
<td>FVC</td>
<td>&gt;80%</td>
<td>60-79%</td>
<td>51-59%</td>
<td>&lt;50%</td>
</tr>
</tbody>
</table>

Another instrument which is used to measure lung function is a peak flow meter. This is a less expensive way of measuring lung function and can also be used in the outpatient management of asthma because it is easier to use and more freely available than spirometers.\textsuperscript{33} A peak flow meter measures peak expiratory flow rate (PEFR) which is the maximum flow obtained during the FVC. The device must initially be reading zero and either in a sitting or standing position the patient must breathe in as far as possible. Thereafter the patient must place the peak flow meter in his/her mouth, closing the lips around the mouthpiece, and must then blow out as hard and as fast as possible for at least two seconds, being sure not to cough or let the tongue obstruct the mouthpiece. The value obtained must be recorded and the process repeated at least twice. The two highest values must be recorded and must be within 10% of each other, with the highest value then being used to assess lung function.\textsuperscript{34} PEFR is less sensitive and more variable and is strongly dependent on how hard the patient tries. It is also dependent on age, sex and body size. Predicted normal spirometric values, including PEFR, are higher for males in comparison with females.\textsuperscript{29} The most common method of relating PEFR to environmental and other variables of interest is to express PEFR as a percentage of a predicted or reference value drawn from a study of a population suitable for this purpose. Readings should be taken at the same time every day, twice a day on awakening and at bed-time. Readings should be charted to establish basal levels and then to monitor large diurnal variations of a progressive decline.\textsuperscript{33} Some patients with asthma are encouraged to record twice daily PEFR measurements, clinical symptoms and $\beta_2$-agonist use in a diary. The early recognition of a deterioration of their PEFR may trigger the
commencement of a course of oral corticosteroids, an increase in their inhaled corticosteroid dose or early presentation to their doctor for clinical assessment. Peak flow meters are more easily available and more patients should be encouraged to use this method of monitoring their disease. As with inhaler devices, the technique used in measuring a PEFR should be checked and reinforced.\textsuperscript{17}

The following tables outline the predicted PEFR values in litres/minute for normal adult males and normal adult females with no airway obstruction:

Table 2.4 Predicted PEFR values in healthy adult males\textsuperscript{21}

<table>
<thead>
<tr>
<th>Normal adult males</th>
<th>Age (Years)</th>
<th>160cm</th>
<th>168cm</th>
<th>175cm</th>
<th>183cm</th>
<th>191cm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>15</td>
<td>600</td>
<td>615</td>
<td>625</td>
<td>635</td>
<td>650</td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>585</td>
<td>600</td>
<td>610</td>
<td>625</td>
<td>635</td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>570</td>
<td>585</td>
<td>595</td>
<td>605</td>
<td>620</td>
</tr>
<tr>
<td></td>
<td>30</td>
<td>555</td>
<td>565</td>
<td>580</td>
<td>585</td>
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<td></td>
<td>35</td>
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<td></td>
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<td>520</td>
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<td>540</td>
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<td>65</td>
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<tr>
<td></td>
<td>70</td>
<td>605</td>
<td>620</td>
<td>635</td>
<td>645</td>
<td>655</td>
</tr>
</tbody>
</table>
Table 2.5  Predicted PEFR values in healthy adult females\textsuperscript{21}

<table>
<thead>
<tr>
<th>Age (Years)</th>
<th>152cm</th>
<th>160cm</th>
<th>168cm</th>
<th>185cm</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>445</td>
<td>455</td>
<td>465</td>
<td>475</td>
</tr>
<tr>
<td>20</td>
<td>460</td>
<td>470</td>
<td>485</td>
<td>495</td>
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<tr>
<td>25</td>
<td>470</td>
<td>485</td>
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<td>30</td>
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<td>40</td>
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<td>45</td>
<td>460</td>
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<td>50</td>
<td>445</td>
<td>455</td>
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<td>55</td>
<td>435</td>
<td>445</td>
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</tr>
<tr>
<td>60</td>
<td>425</td>
<td>430</td>
<td>440</td>
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<td>65</td>
<td>410</td>
<td>420</td>
<td>425</td>
<td>435</td>
</tr>
<tr>
<td>70</td>
<td>395</td>
<td>405</td>
<td>415</td>
<td>425</td>
</tr>
</tbody>
</table>

Unfortunately, measurements of lung function have never become established in general practice because of cost, inconvenience, and misconceptions about the difficulty and complexity of spirometry. To counter these factors, industry has begun to develop simple-to-use, handheld spirometers for physicians’ offices and clinics.\textsuperscript{29}

The measurements used when assessing asthma symptom severity normally include PEFR, spirometry measurements such as FVC, FEV\textsubscript{1}, forced expiratory flow during 25-75% FVC (FEF\textsubscript{25-75}), daytime symptoms such as wheeze, coughing or a tight chest and night time symptoms such as wheeze, cough, tightness of the chest and night waking.\textsuperscript{4,5} A study by Urek \textit{et al} (2004)\textsuperscript{35} found that PEFR was an especially good indicator of asthma activity and this suggestion has been reinforced by a number of other studies.\textsuperscript{36-38} Asthma severity can therefore be assessed quite accurately and easily at home if the patient has access to a peak flow meter. No complicated spirometry measurements are needed. However, most patients in the primary health care setting will not be able to acquire their own peak flow meters due to the generally high cost of this equipment.
2.3.2. QUESTIONNAIRES USED TO ASSESS ASTHMA

Traditionally, clinicians and clinical investigators have used conventional methods of measuring asthma such as airway calibre spirometry, symptoms, use of ß agonists, and responsiveness of the airways to cold air and exercise as components of the comprehensive assessment of the patient’s asthma status. However, none of these parameters actually indicate whether the patient is able to function better in their day-to-day life. It has been assumed that if one of these indices improves, the patient’s health-related quality of life must also have improved, however this may not always be the case. More sensitive measures are needed to capture subtle changes in asthma management. Two types of measures are widely used to assess health-related quality of life (QOL) namely generic QOL instruments that are standardized and applied widely to enable comparisons between patients with different types of illness and disease specific QOL instruments for more intensive research on a particular condition. Consequently, disease-specific quality of life measures for asthma have been developed and show promise for asthma research.

Examples of some of the questionnaires developed for assessing health-related quality of life in asthmatic patients include the St George’s Questionnaire and The Hyland’s living with Asthma Questionnaire. Juniper et al (1999) developed an Asthma Quality of Life Questionnaire (AQLQ). This questionnaire covers the main spheres of human living, which includes work place and home assignments, sleeping and relaxation, sport and recreation, family affairs, social contacts, health care, and material status. It also allows for the identification and inclusion of five patient-specific activities for example playing with pets. It has strong measurement properties and has been validated for use in both clinical trials and clinical practice and can be used to assess patient quality of life pre- and post-intervention. It also has good discriminative properties which mean that in surveys, it is able to detect differences between patients who have different levels of asthma quality of life. In addition, it has strong evaluative properties and it is therefore sensitive to change and can detect small but important changes that patients experience either as a result of an intervention or natural fluctuation in the condition. A short form of this questionnaire, namely the AQLO(S) has also been validated by Juniper et al as having the same measuring properties as the original AQLQ however it is more practical to use since it does not
contain the five patient-specific activities which have been proven to change over time. In the AQLQ(S) a predetermined generic list of activities is provided and this problem has therefore been eliminated. This questionnaire is more suitable for use in measuring the quality of life of asthma patients since it is a disease-specific questionnaire and not a generic questionnaire. For the AQLQ, the difference or change in score that can be considered clinically meaningful has been estimated. This is the Minimal Important Difference (MID) which has been defined as “the smallest difference in score which patients perceive as beneficial and would mandate, in the absence of troublesome side-effects and excessive cost, a change in the patient’s management”. The MID for all versions of the AQLQ has been estimated to be close to 0.5 on the seven-point scale. A study by Xuan et al (1999) suggests that co-morbid conditions significantly and extensively affect patients’ scores on generic QOL measures and estimation of treatment effect, whereas their influence on disease specific QOL scores and estimation of treatment effect is considerably smaller.

A number of questionnaires are available to measure asthma patient knowledge about their disease. Some examples of questionnaires include: the Asthma Knowledge Test, the Asthma General Knowledge Questionnaire for Adults (AGKQA) and the Knowledge Attitude and Self-efficacy Asthma Questionnaire (KASE-AQ). This KASE-AQ questionnaire developed by Wigal et al (1993) is especially useful since it measures all three elements of asthma knowledge, namely their knowledge regarding asthma, their attitudes regarding their asthma (including their willingness to cooperate with their physician in managing asthma) and their self-efficacy regarding their perceived ability to control the disorder. These changes in patient variables are assessed following a particular intervention. The KASE-AQ has been demonstrated to be reliable and internally consistent.
2.7 ASSESSMENT AND CLASSIFICATION OF ASTHMA IN SOUTH AFRICA

In South Africa, asthma is assessed on presentation as mild intermittent or chronic persistent asthma. Chronic persistent asthma can be further categorised as mild, moderate or severe. When in doubt the patient should be placed in a more severe category. This assessment system is of less value in patients who are already on treatment, but provides a guide to the level of control and the need for additional treatment. Following assessment of severity, the appropriate level of initial treatment is selected.²
A summary of the assessment of asthma severity according to The South African Guidelines for the Management of Chronic Asthma in Adults is indicated in Table 2.6.

### Table 2.6  Assessment of asthma severity at presentation using symptoms (daytime cough, tight chest and wheeze), night waking due to symptoms and lung function (PEF - peak expiratory flow)*2

<table>
<thead>
<tr>
<th>INTERRMITTENT</th>
<th>CHRONIC PERSISTENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>Mild</td>
</tr>
<tr>
<td>I</td>
<td>II</td>
</tr>
</tbody>
</table>

**DAYTIME SYMPTOMS:**
- any of cough, tight chest, wheeze
- ≤2/week

**NIGHT SYMPTOMS**: *
- ≤1/month

**PEF**
- ≥ 80%

---

**CHRONIC PERSISTENT**
- Moderate
- Severe
- III
- IV

**DAYTIME SYMPTOMS:**
- any of cough, tight chest, wheeze
- 2-4/week

**DAYTIME SYMPTOMS:**
- any of cough, tight chest, wheeze
- >4/week

**DAYTIME SYMPTOMS:**
- any of cough, tight chest, wheeze
- continuous

**NIGHT SYMPTOMS**
- 2-4/month

**NIGHT SYMPTOMS**
- >4/month

**NIGHT SYMPTOMS**
- frequent

**PEF**
- ≥80%

**PEF**
- 60-80%

**PEF**
- <60%

* presence of any one or more of daytime symptoms, night symptoms and PEF defines severity category. If in doubt place in the more severe category.

** includes wheeze, cough, tightness of the chest and night waking
2.5 TREATMENT OF ASTHMA

The knowledge that inflammation plays a primary role in the pathogenesis of asthma has led to the conviction that the focus of therapy is the prevention and suppression of the underlying inflammation. Thus current therapeutic options in asthma consist of acute reliever medications (relievers) used for acute exacerbations and long-term control medications used for the prevention (preventers) of symptoms and the suppression of inflammation. The currently accepted approach is to use drugs that suppress the inflammatory response as primary long-term control therapy, thereby reducing the degree of bronchial hyperresponsiveness (BHR) and improving long-term control and outcomes in asthma by preventing airway remodelling.

The following classes of drugs are used in the management of asthma: β2 agonists, methylxanthines, anticholinergics, sodium cromoglycate, corticosteroids, leukotriene receptor antagonists and various other agents.

Table 2.7 illustrates the classes of drugs, as divided into preventers, controllers and relievers.

Table 2.7 Classification of drugs*

<table>
<thead>
<tr>
<th>PREVENTERS</th>
<th>CONTROLLERS</th>
<th>RELIEVERS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inhaled corticosteroids</td>
<td>Long-acting β2 agonists</td>
<td>Short-acting β2 agonists</td>
</tr>
<tr>
<td>Oral corticosteroids</td>
<td>Sustained-release theophylline tablets</td>
<td>Anticholinergics</td>
</tr>
<tr>
<td></td>
<td>Leukotriene antagonists</td>
<td>Short-acting theophyllines</td>
</tr>
</tbody>
</table>

*Adapted from the Guidelines for the Management of Chronic Asthma in Adults 2000
2.5.2  ß2 AGONISTS

The ß2 agonists are the most effective bronchodilators available.3 They act by activating ß2 receptor subtypes resulting in activation of adenylyl cyclase and increased conversion of adenosine triphosphate (ATP) to adenosine 3’5’-cyclic monophosphate (cAMP). Activation of the cyclase enzyme is mediated by the stimulatory coupling protein Gs. The major second messenger of ß-receptor activation is cAMP.46

Besides enhancing bronchoselectivity, the aerosol administration of ß2 agonists provides greater protection than systemic administration against provocations that induce bronchospasm such as exercise and allergens.47 Currently, the only disadvantage of aerosol administration of ß2 agonists is the complexity of administration.3 They can however also be administered by the oral or parenteral routes. Oral ß2 agonists should only be prescribed for patients who cannot manage the inhaled route, which should be a very small minority.48 Intravenous ß2 agonists are used in severe asthma and possess bronchodilator potency comparable to that of aminophylline.48 The long-acting inhaled ß2 agonists, salmeterol and formoterol, are lipid-soluble and provide long-lasting bronchodilation of twelve hours or more.

Inhaled short-acting selective ß2 agonists are indicated for the treatment of intermittent episodes of bronchospasm and are the treatment of choice for acute severe asthma.47 Long-acting inhaled ß2 agonists are indicated as adjunctive long-term control for patients with symptoms who are already on low to medium doses of ICS prior to advancing to medium-or high dose ICS.4 The regular use of short-acting inhaled ß2 agonists does not completely prevent symptom onset and the frequency of their use may therefore indicate the level of asthma control, so it is recommended that they only be used as needed to control asthma symptoms.4

Chronic administration of ß2 agonists can lead to a down-regulation of ß2 receptors and a decreased binding affinity for these receptors. This results in tolerance to the effect of the drug. Concurrent systemic corticosteroid therapy can both prevent and partially reverse this phenomenon.47 Skeletal muscle tremor is a common side effect associated with ß2 agonist use. At high doses ß2 agonists also have ß1 effects thus
cardiac effects such as tachycardia are common even with inhalation therapy and excessive use may lead to arrhythmias.\textsuperscript{46}

Examples of $\beta_2$ agonists available in South Africa include: salbutamol, fenoterol and terbutaline which are all short-acting, and salmeterol and formoterol which are long-acting agents. Formoterol can however be used during an acute attack since it has a more rapid onset of action and fewer side effects at more frequent dosing intervals. Hexoprenaline is also a selective $\beta_2$ agonist for systemic use.\textsuperscript{48} Salbutamol inhalers are available in the public health care setting in South Africa. Long-acting $\beta_2$ agonists are not available on the EDL.

Salbutamol, fenoterol and hexoprenaline interact with $\beta$ blockers such as propranolol, as well as digoxin, ipratropium bromide and methyldopa. The concurrent use of $\beta_2$ agonists and theophylline, corticosteroids or potassium-depleting diuretics can lead to hypokalemia resulting in heart arrhythmias.\textsuperscript{49}

\subsection*{2.5.2 METHYLXANTHINES}

Methylxanthines inhibit phosphodiesterase (PDE), the enzyme that metabolises cAMP to AMP and thereby cause an increase in cAMP which causes bronchodilation.\textsuperscript{46}

Theophylline is the most common methylxanthine used in the treatment of asthma and is indicated for the second-line treatment of acute, severe and chronic persistent asthma.\textsuperscript{3} It has a very narrow therapeutic range (5-15$\mu$g/mL)\textsuperscript{3} and close therapeutic drug monitoring is therefore needed since theophylline intoxication can result in severe cardiac effects, seizures and death.\textsuperscript{20} Dose-dependent side effects that can be expected with theophylline use include nausea, gastrointestinal upset, headache, arrhythmias, tachycardia and convulsions.\textsuperscript{49}

Drug interactions with theophylline are common. Its plasma concentration is decreased by drugs that induce P450 enzymes such as rifampicin, phenobarbitone, phenytoin and carbamazepine. Drugs which inhibit P450 enzymes such as oral contraceptives, erythromycin, ciprofloxacin and calcium-channel blockers will lead to an increased serum theophylline concentration\textsuperscript{40} while beta-blockers may reduce the
clearance of theophylline. On the other hand rifampicin and phenytoin may lower the serum levels of theophylline and thyroid compounds may lead to an increase in theophylline requirements. Theophylline interacts with β-agonist bronchodilators which can lead to serious hypokalemia resulting in arrhythmias.

Aminophylline is another methylxanthine available for the treatment of asthma and is only administered parenterally. Theophylline is the only methylxanthine available on the Essential Drugs List (EDL) as a sustained release 250mg tablet.

2.5.3  ANTICHOLINERGICS

Ipratropium bromide is an antimuscarinic agent which, when given as an aerosol, competitively blocks muscarinic receptors in the airways resulting in bronchodilation. In South Africa it is most commonly used for COPD since it seems to be most beneficial in these patients. It is available on the EDL in South Africa and is indicated for the relief of bronchospasm in reversible airways obstruction. It is not commonly used alone in asthma.

Side effects which can be expected with the use of ipratropium bromide include a dry mouth and a bitter taste. Since it is given by inhalation, systemic antimuscarinic effects such as urinary retention and constipation are rare. Concomitant use of ipratropium bromide and salbutamol as a nebulising solution can lead to acute glaucoma in susceptible patients.

Tiotropium bromide is a newer antimuscarinic drug used for COPD in South Africa. It is not available on the EDL.

2.6.4  SODIUM CHROMOGLYCATE

Sodium chromoglycate (cromolyn) is a mast cell stabiliser and is indicated for the treatment of asthma in children. It is also used for allergic rhinitis and this is the condition it is most frequently used for in South Africa. It is not available on the EDL.
2.6.5 CORTICOSTEROIDS

Research shows that corticosteroids are the most effective anti-inflammatories available to treat asthma. They are useful in the treatment of asthma since they increase the number of β2 adrenergic receptors and enhance the receptor responsiveness to β2 adrenergic stimulation. They also reduce mucous production and hypersecretion as well as BHR and prevent and reverse airway remodelling. Most importantly, they are inflammatory response mediators. Corticosteroids are highly lipophylic, thereby readily crossing the cell membrane and combining with the glucocorticoid receptor. This activated complex then enters the nucleus where it causes gene activation or suppression. This leads to specific mRNA production, resulting in increased production of anti-inflammatory mediators such as lipocortin-1, neutral endopeptidase and endonucleases. Less pro-inflammatory cytokines such as interleukin-1 (IL-1) and granulocyte-macrophage colony stimulating factor (GM-CSF) are also synthesized and released, reducing inflammatory cell activation, recruitment and infiltration and decreasing vascular permeability.3

Corticosteroids used in the management of asthma are available in oral, inhaled and parenteral dosage forms. The inhaled route is preferred for the management of asthma since it does not exhibit the potential range of side-effects to the same extent that the oral and parenteral routes do. Systemic corticosteroids should therefore be reserved for cases where other therapies are unsuccessful or to prevent an impending episode of severe asthma. Because short term (1-2 weeks) high-dose corticosteroids (1-2mg/kg/d) do not produce serious toxicities, the ideal regimen is to administer systemic corticosteroids in a short burst and then to maintain the patient on other treatments, including inhaled corticosteroids (ICSs), with long periods between systemic corticosteroid treatment. If chronic systemic corticosteroid therapy is needed to control a patient’s asthma, the lowest possible effective dose should be used. Often complete symptom control has to be sacrificed to minimise toxicity of corticosteroids. Side-effects can be minimised by using alternate-day therapy or by using high-dose ICSs.3

Adverse effects that can be expected with chronic systemic glucocorticoid administration include osteoporosis, sodium and water retention, hyperglycaemia,
hypokalemia, hypertension, skin striae, glaucoma and impaired wound healing. ICSs can result in adrenal suppression but this is rarely significant. Side-effects from ICSs include oropharyngeal candidiasis as well as laryngeal myopathy, hoarseness and a sore throat all of which can be minimised with the use of a spacer and by rinsing the mouth with water after inhalation.

Patients should be advised of the necessity to continue ICS use even if asthma symptoms disappear and should be counselled on the preventative nature of corticosteroid therapy.

Inhaled corticosteroids available in South Africa include beclomethasone, budesonide and fluticasone. Beclomethasone and budesonide are listed on the EDL. Systemic corticosteroids available in South Africa include hydrocortisone sodium succinate, methylprednisolone, dexamethasone, prednisone and prednisolone as well as betamethasone. All of these agents are available on the EDL, however prednisone and hydrocortisone are recommended for use in acute asthma according to the Standard Treatment Guidelines and EDL for adults at a hospital level.

2.6.6 LEUKOTRIENE RECEPTOR ANTAGONISTS

Leukotrienes released during the inflammatory process cause contraction of smooth muscle, microvascular leakage and sputum secretion, and attract eosinophils. Montelukast and zafirlukast inhibit the effect of leukotrienes at the receptor level. Zileuton interrupts the conversion of arachidonic acid into leukotrienes. The leukotriene receptor antagonists exhibit both bronchodilator and anti-inflammatory activity. The exact place of these agents in the management of asthma is still being clarified. Montelukast and zafirlukast are particularly valuable as supplements to inhaled corticosteroids in patients who remain symptomatic and patients with asthma associated with aspirin and non-steroidal anti-inflammatory drug (NSAID) intolerance seem to be more responsive. Zileuton is effective in preventing both exercise and antigen-induced bronchospasm. Leukotriene receptor antagonists are however not indicated for the treatment of acute asthma and should rather be used as preventers.
Side effects of the leukotriene receptor antagonists include abdominal pain, dyspepsia, dizziness, headache, diarrhoea, cough and fever as well as hypersensitivity reactions and eosinophilia. Zafirlukast and zileuton have also been associated with elevated liver enzymes.\textsuperscript{48} Zafirlukast is extensively metabolised by the cytochrome P450 system and its levels can be reduced by the concomitant use of theophylline.\textsuperscript{48}

None of the leukotriene receptor antagonists are as yet available on the EDL.
2.7 ASTHMA MANAGEMENT

The NAEPP has set the following goals for asthma management:4

1. Maintain normal activity levels (including exercise and other physical activity).
2. Maintain (near) normal pulmonary functions.
3. Prevent chronic and troublesome symptoms (for example coughing or breathlessness in the night, in the early morning or after exertion).
4. Prevent recurrent exacerbations of asthma and minimise the need for emergency department visits or hospitalisations.
5. Provide optimal pharmacotherapy with minimal or no adverse effects.
6. Meet patients’ and families’ expectations of satisfaction with asthma care.

In patients with known allergic triggers for their asthma, allergen avoidance has resulted in improvement of their symptoms, a reduction in medication use and a decrease in BHR. Simple environmental controls for patients with house dust mite allergy include removing carpeting from bedrooms, washing sheets in hot water (>55ºC) and using plastic pillow and mattress covers.51 Obvious environmental triggers, such as cockroaches, should be avoided if the patient is sensitive but there is little evidence that extensive environmental controls such as home air-filtering devices are of any value.4 Smokers should be encouraged to stop since smoking is one of the contributing factors to asthma.20
In South Africa, the following guidelines are used for the management of chronic asthma in adults as published by the Allergy Society of South Africa (ALLSA).²

Figure 2.3  Guidelines for the management of chronic asthma in adults²

A stepwise approach is followed whereby the patient’s asthma severity is firstly assessed and treatment is then initiated according to the classification of the asthma severity. The principle of “hit early, hit hard” is used whereby the patient will be
placed in the more severe category if some of the symptoms overlap between two categories.\textsuperscript{2} Treatment can be stepped down a category if symptoms control is achieved for three months and the use of oral corticosteroids should be stopped or reduced first. A pulse course of oral corticosteroids (30-40mg per day for seven to fourteen days) can be given at any time if symptoms become uncontrolled.
The Essential Drugs List for use at a primary health care level in South Africa also presents guidelines for the management of asthma.13

Table 2.8  EDL guideline for the management of asthma13

<table>
<thead>
<tr>
<th>Intermittent asthma</th>
<th>Mild persistent asthma</th>
<th>Moderate persistent asthma</th>
<th>Severe persistent asthma</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Not more than one or two episodes of daytime cough and/or wheeze per week</td>
<td>• 2-4 episodes of wheeze and/or cough per week</td>
<td>• More than 4 episodes of daytime wheeze, tightness or cough per week</td>
<td>• Continuous wheeze, tightness, cough</td>
</tr>
<tr>
<td>• Less than one night-time cough and/or wheeze per month</td>
<td>• 2-4 episodes of night time wheeze or cough</td>
<td>• More than 4 night time awakenings per month</td>
<td>• Frequent nocturnal symptoms</td>
</tr>
<tr>
<td>• No recent (within the last year) admission to hospital for asthma</td>
<td>• PEFR more than 80% predicted between attacks</td>
<td>• PEFR more than 60% but less than 80% predicted</td>
<td>• PEFR less than 60% predicted</td>
</tr>
<tr>
<td>• β₂ agonist inhalation, 1-2 puffs 3-4 times daily as needed until symptoms are relieved</td>
<td>• Corticosteroid inhalation, low dose, 12 hourly regularly</td>
<td>• Corticosteroid inhalation, medium dose, 12 hourly regularly</td>
<td>• Refer patient for review of condition</td>
</tr>
<tr>
<td></td>
<td>• β₂ agonist inhalation, 1-2 puffs 3-4 times daily as needed until symptoms are relieved</td>
<td>• β₂ agonist inhalation, 1-2 puffs 3-4 times daily as needed until symptoms are relieved</td>
<td></td>
</tr>
</tbody>
</table>

The EDL guidelines do not include theophylline in the management of asthma whereas the ALLSA guidelines recommend theophylline treatment in the management of mild persistent, moderate persistent and severe persistent asthma. The EDL only recommends theophylline for the treatment of COPD. Both the EDL and
ALLSA guidelines recommend inhaled corticosteroids in the management of mild persistent, moderate persistent and severe persistent asthma and the ALLSA guidelines also recommend the use of inhaled corticosteroids in the management of intermittent asthma at a lower dose. Inhaled β2 agonists are recommended by both the EDL and the ALLSA guidelines for the management of all severity levels of asthma.²,¹³

Exacerbations of asthma usually occur gradually over several days to weeks or on a background of chronic poor asthma control. This provides an opportunity for early intervention with corticosteroids and β₂ agonists which act to reverse airflow obstruction and reduce the severity of the exacerbation. A written action plan facilitates the early detection and treatment of an exacerbation and is therefore an essential part of the self-management of exacerbations.⁵² An action plan is a set of instructions provided to a patient with asthma for use in the management of deteriorating asthma. An individualised written action plan is tailored to the patient’s underlying asthma severity and treatment. It is further characterised by being a written plan which informs the subject about when and how to modify medications and how to access the medical system in response to worsening asthma.⁵²


2.7 USE OF INHALERS

Nearly 40% of all patients use their inhalers incorrectly but treatment failure is often blamed on the medication.\textsuperscript{53} When their condition does not improve, patients may think the medication is not working when, in reality, they are not achieving maximal lung access.\textsuperscript{53} Different types of devices deliver asthma medication to the lungs with varying efficacy.

The most important device factor determining the site of aerosol deposition is particle size. Particles larger than 10µm get deposited in the oropharynx, particles between 5 and 10µm deposit in the trachea and large bronchi, particles 1 to 5µm reach the lower airways, and particles smaller than 0.5µm act as a gas and are exhaled.\textsuperscript{3} In the treatment of asthma, the airways, not the alveoli, are the target for drug delivery. The most important patient factor determining aerosol deposition is inspiratory flow. The degree of deposition will be increased by high inspiratory flows due to the impaction of particles, thereby increasing central deposition (in the large airways) and decreasing peripheral deposition.\textsuperscript{3}

Inhaled asthma medication is divided into three groups: relievers, preventers and controllers.\textsuperscript{17} Relievers are generally packaged in blue containers while preventers are normally packaged in brown or orange containers. Controllers normally come in green packaging. Relievers are short-acting, quick-onset bronchodilators such as salbutamol which produce relief from the symptoms of asthma. They are normally used on an "as required" basis. The drugs contained in these formulations do not treat the underlying inflammation. Preventers consist of drugs which act on the underlying inflammation associated with asthma. These drugs are normally corticosteroid based such as inhaled corticosteroids and are required to be taken regularly twice a day, even when the patient feels well. Controllers include those drugs which are long-acting such as salmeterol and formoterol and have a slow onset of action. They have a sustained bronchodilator action and some of these drugs may have some anti-inflammatory action.

Inhaled corticosteroids (ICSs) are the cornerstone of modern asthma treatment.\textsuperscript{1} As asthma severity increases the dose of ICSs is stepped up and other classes of drugs are
added, particularly long-acting β₂ agonists (see guidelines section 2.6). Once control of asthma has been achieved and maintained for at least three months, a gradual reduction of the maintenance therapy should be tried in order to identify the minimum therapy needed to maintain control. However, there is evidence that not all asthma patients who require ICSs receive them.54

### 2.7.1 Metered Dose Inhalers

Pressurised metered dose inhalers (MDIs) have been used to deliver asthma medication to the lungs for almost half a century. However, they have many disadvantages both in terms of effectiveness and usability.55 They are inefficient, typically delivering only about a third of the emitted dose to the lungs and less than half of the emitted dose to the peripheral airways. They also require good coordination of inhaler activation and inspiration to ensure correct inhalation and deposition of drug in the bronchial tree.56 Metered dose inhalers are the only type of inhalers available for the treatment of asthma in the primary health care setting.

The MDI is a small aerosol canister in a plastic holder. They usually contain active drug, low-vapour-pressure propellants such as chlorofluorocarbon (CFC) or hydrofluoroalkane (HFA), cosolvents and/or surfactants.³ By pressing down on the canister, a propellant forces a pre-measured burst of medication, consisting of a cloud of particles with a mass median aerodynamic diameter (MMAD) of 45µm, out of the canister and into the mouth, which can be breathed in deeply to ensure deposition in the lungs.53 As evaporation occurs, the particle size is reduced to a final MMAD of 2.8 to 5.5µm, depending on the MDI.³

A six-step scale developed by Williams et al (1998)⁵⁷ can be used to assess patients’ ability to correctly use a MDI namely: (1) remove the MDI cap and shake the inhaler, (2) exhale slowly prior to inhalation, (3) actuate the MDI at onset of inhalation, (4) inhale at less than maximal rate, (5) hold breath after inhalation for at least 5 seconds, and (6) wait at least 30 seconds between each MDI actuation. Even with a good technique, only about 10% of the emitted dose actually reaches the lungs. When the inhaler is used incorrectly, an even smaller amount may reach the lungs.⁵³ Patients frequently fail to exhale fully before the inhalation and to continuously inhale slowly.
after activation of the inhaler. In addition, patients often activate the inhaler before inhalation or at the end of inhalation and conclude inhaler activation while breath-holding.\textsuperscript{58}

With optimal technique about 10\% to 30\% of the metered dose is deposited in the lung, depending on the device. About 70\% to 80\% impacts on the oropharynx due to the high initial velocity and this portion is then swallowed. The rest is either left in the actuator or exhaled.\textsuperscript{3} The newer hydrofluoroalkane (HFA)-propelled beclomethasone dipropionate MDI is a marked exception, delivering 50\% to 60\% of its actuated dose to the lungs with optimal technique.\textsuperscript{3} It also has a less forceful spray with smaller particles.

Gayrard and Orehek (1980)\textsuperscript{59} assessed MDI use in a group of 115 asthmatics. Patients were divided into two groups. The first group received instruction on the correct use of their MDI by a physician and the need for correct inhaler use was strongly emphasised. The second group received no instruction and used their inhaler according to manufacturers’ instructions. Of patients who received no instruction, 72\% were unable to use their MDI correctly compared with 48\% following training by a physician. A Spanish study with 1640 volunteers (746 patients, 466 nurses, 428 physicians) showed that 91\% of patients were unable to use an MDI correctly compared with 85\% of nurses and 72\% of physicians. General practitioners and paediatricians had the worst inhaler technique when compared with pulmonologists and allergists.\textsuperscript{60} This is especially important when one looks at the primary health care setting where a general practitioner is generally the most qualified person who will see an asthma patient before medication is initiated.

A study by Williams \textit{et al} (1998)\textsuperscript{57} determined the relationship of literacy to asthma knowledge and ability to use a MDI among patients with asthma. Patients with poor reading skills had significantly less asthma knowledge and poorer MDI skills. In a multivariate analysis, patients’ reading level was the strongest predictor of asthma knowledge and MDI skills. Although MDI skills were better amongst the more literate patients, they were still inadequate. The results of this study confirm that patients with poor reading skills do not fully comprehend medical instruction using standard patient education methods. The researchers suggested that patients with low literacy should
be involved in developing educational materials to empower them to improve their health while ensuring effective educational content and that successful education programs, that reduce morbidity and mortality of those most affected by asthma, must consider patients’ literacy skills. Again, this particular study is of great importance in the primary health care setting where patients often have inadequate literacy skills.

Misuse of MDIs has been shown to be the rule rather than the exception, mainly due to poor coordination, and this correlates with poorer asthma control in asthmatic patients treated with ICS.55 Inability to correctly use inhaler devices is a major reason for non-compliance with therapy. Patients need to be completely informed of the correct use of the device and must be observed using the device in order to assess inhalation technique. Although clinicians have noted problems with patients’ ability to use MDIs for many years, published data demonstrating improved outcome in the traditional clinic setting are scarce. Although correct MDI usage requires adherence to a seemingly simple set of instructions, a significant proportion of patients find these instructions confusing and therefore fail to use their inhalers correctly.56 Correct use of MDIs thus requires intensive training and regular technique re-testing.

2.7.2 SPACERS

A device called a spacer is highly recommended to make the MDI easier and more effective to use. The purpose of a spacer is to allow evaporation of the propellant prior to inhalation. Additionally, most of the large particles that would normally deposit in the oropharynx rain out in the spacer.3 A spacer is simply a clear plastic bottle-like container with a mouthpiece at one end and a hole at the other into which an inhaler can be fitted. The spacer may also include a one-way valve near the mouthpiece end that allows air to be sucked out of the spacer but prevents air from being blown into it. Several advantages of spacer use have been reported. These include less skill and coordination required for its use, an increase in the dose of medication reaching the lungs as well as a decrease in droplet size with the use of large volume spacers16. The spacer extends the space between the inhaler mouthpiece and the patient’s mouth. This allows the force of spray to diminish during the inhalation and the patient has additional time to begin inhaling after release of the medication. Spacers also collect
the larger droplets of inhaled medication so that patients only inhale the smaller particles that are more easily respirable.\cite{61}

All the available spacers significantly reduce oropharyngeal deposition of the CFC-propelled aerosols. The lung delivery through a spacer depends on both the MDI and the drug, where one device may enhance delivery with one MDI preparation and decrease delivery with others.\cite{3}

A study by Zar \textit{et al} (1999)\cite{62} showed that a 500mL plastic bottle and a conventional spacer yielded a similar response to a $\beta_2$ agonist given via MDI in children with acute asthma. The bottle was modified carefully to create a good seal between the MDI and the hole in the base of the bottle. A heated wire mould of the same shape and size as the MDI was applied to the base of the plastic bottle to melt the plastic and make a hole. The MDI canister was then inserted immediately, to create a tight fit. Plastic bottles were primed with 15 puffs of bronchodilator to reduce the electrostatic charge of the sidewalls and to optimise drug delivery. Electrostatic charge can also be reduced by rinsing the bottles in detergent. Single rather than multiple actuations of the MDI also ensured maximum drug delivery. The bottle is limited as a spacer because it has no one-way valve. Exhaled air may therefore enter the spacer, to dilute the aerosol inside the spacer. However, the study results suggest that, in practice, absence of a valve does not adversely affect the response to bronchodilator.\cite{62}

Besides assisting in the delivery of medication to the lungs, spacers help reduce the side effects that may result from some inhaled medications.\cite{53} Medicine inhaled directly from the inhaler, travelling at such a high speed, tends to hit the back of the throat or other areas of the mouth. A corticosteroid medication that is deposited in the throat may cause hoarseness, sore throat, or oral candidiasis.\cite{16} Spacers increase the amount of drug being deposited in the lungs thereby decreasing the amount of drug deposited in the mouth and throat. This reduces the incidence of the abovementioned oral side effects.\cite{16}

2.7.3 \textbf{BREATH-ACTUATED INHALERS}

Breath-actuated inhalers deliver medication automatically when a slow, deep breath is taken. The breath-actuated inhaler device is cocked with a lever to load the dose of
medication, a baffle is opened by inspiratory pressure and the dose is expelled from the canister metering chamber.\textsuperscript{3} There is no need to press anything to release the drug, and a spacer is not necessary.\textsuperscript{53} Types of breath-actuated inhalers include the Accuhaler\textsuperscript{®}, Diskhaler\textsuperscript{®}, Rotahaler\textsuperscript{®}, Spinhaler\textsuperscript{®} and Turbuhaler\textsuperscript{®}.\textsuperscript{20} Breath-actuated inhalers are not available in the primary health care setting in South Africa due to cost factors. These devices improve pulmonary drug deposition only in patients unable to coordinate the use of conventional MDIs adequately and may be particularly useful in the elderly who normally have difficulty actuating conventional MDIs.\textsuperscript{3}

Dry-powder inhalers (DPIs) are a special type of breath-actuated inhalers and rely solely on the force created by the patient’s inspiration to draw the medication into the lungs. This means that a high inspiratory flow will ensure good drug delivery in the bronchioles. DPIs do not contain propellants or any other ingredients besides the medication.\textsuperscript{53} There are various types of DPIs, each with different operating methods. Examples of DPIs available in South Africa include Accuhaler\textsuperscript{®}, Turbuhaler\textsuperscript{®}, Diskhaler\textsuperscript{®} and Autohaler\textsuperscript{®}.\textsuperscript{48} In general, DPIs require higher inspiratory flows (>60L/min) and a change in inhalation technique (deep, forceful inspiration) for optimal actuation as compared with MDIs, which in turn increase the amount of drug delivered to the larger central airways.\textsuperscript{3} Common to all DPIs is the fact that desagglomeration of the powdered medication is caused by the inspiratory flow generated by patients which makes drug delivery inspiratory flow-dependent. DPIs offer a number of advantages to conventional MDIs without necessarily increasing costs. One of their main advantages is that inhaler activation and inhalation of the aerosol requires no coordination by the patient.\textsuperscript{55}

### 2.7.4 NEBULISERS

A nebuliser changes asthma medication from a liquid to a mist so that it can be more easily inhaled into the lungs. Two basic forms of nebulisers are available namely jet nebulisers and ultrasonic nebulisers. Jet nebulisers produce an aerosol from a liquid solution or suspension in a cup. A baffle creates a droplet cloud that is inhaled. Ultrasonic nebulisers produce an aerosol by vibrating liquid lying above a transducer at speeds of about 1mHz. The MMAD of the droplets is directly related to the gas flow, with flow of 5 to 12L/min providing an aerosol cloud with a MMAD of 4 to
8µm for most nebulisers. Approximately 10% of the dose placed in a nebuliser is delivered to the patient’s lungs, with 60% to 80% lost in the apparatus, up to 20% exhaled and 2% deposited in the mouth under usual operating conditions. Use of a facemask when compared with a mouthpiece significantly reduces lung delivery due to nasal filtering.³

Home nebuliser therapy is particularly effective in delivering asthma medications to infants and small children and to anyone who is unable to use an inhaler with a spacer.⁵³ Although nebuliser therapy is predominant in the treatment of acute asthma, a pressurised MDI with attached spacer can produce the same or better bronchodilation than a nebuliser even in the presence of severe airways obstruction.⁶³ Because of relative efficiency, ease of use, and low cost, an MDI with spacer may be preferable to a nebuliser for delivery of bronchodilators in acute asthma.⁶³
Optimal management of a chronic disease like asthma requires the active participation of patients. Many of the recommended components of asthma care and management might not be effective without adequate patient education.\(^4\)

Numerous studies demonstrate a marked improvement in asthma management and control with patient education. De Oliveira \textit{et al} (1999)\(^64\) found that an educational programme led to a significant improvement in asthma morbidity and that the implementation of educational programmes is possible for special populations when these programmes are adapted to the socio-economic profile of the patients, with a significant gain in terms of the reduction of symptoms and improved pulmonary function and quality of life of asthmatics. A study by Abdulwabud \textit{et al} (1999)\(^65\) showed that a limited asthma education programme in a hospital outpatient setting had a positive impact on patients’ knowledge of asthma, but not on their quality of life, self-management skills, or attitudes and beliefs about asthma. Interestingly, in the same study it was noted that in the absence of any intervention, the control group increased their asthma knowledge, total quality of life and self-management skills for slow onset asthma attacks. This finding is consistent with the report by Snyder \textit{et al} (1987)\(^66\) that “waiting list controls increased their knowledge of asthma without any intervention other than the periodic filling out of questionnaires.” In their opinion, “asking an asthmatic about asthma improves his awareness and, correspondingly, his understanding of the disorder”. Magar \textit{et al} (2005)\(^67\) also found that control patients received better follow-up than what was normal although they receive minimal education (for example how to take medication properly or how to avoid triggers). It also encourages them to be on their best behaviour so as to be ‘as good as’ their more ‘educated’ counterparts.

Although patient education is not always shown to be effective in changing patient behaviour or outcomes, systematic reviews suggest that patient education for asthma self-management is effective in improving objective measures of lung function, frequency of asthma symptoms and health care utilization.\(^68\)
Goals of asthma education, according to the Guidelines for the Management of Chronic Asthma in South Africa, include:²

- An explanation of the nature of asthma and its allergic basis
- A description of the different classes of drugs and their purpose in treatment
- Advice on prevention strategies (allergens and tobacco smoke avoidance)
- Instruction on the correct use of inhalers and the opportunity to practice under supervision
- Guidelines on how to recognise worsening asthma
- In some patients, particularly those requiring stabilisation or patients who have had a recent exacerbation or deterioration, the use of a PEFR meter and chart
- Introduction to the National Asthma Education Programme (NAEP) – the official asthma education programme of the South African Pulmonology Society.

Patients should also be given instructions on self-management and these include, according to Laloo et al.:²

- Written instructions which should include the class, name, strength, dose and frequency of each of the asthma medications prescribed, including the correct use of inhaler devices
- Provision of realistic goals of treatment in terms of symptom relief and PEFR
- Information on potential side-effects of drugs
- Instruction for patients who may require short courses of oral prednisone on when and how to initiate a course
- Details on when and how to obtain access to medical care in emergencies
- Arrangements for a Medic-Alert badge for patients with severe steroid-dependent asthma, known drug hypersensitivities (like aspirin and penicillin) and brittle asthma
• Advice on how to recognise changes in the asthma and when to make adjustments to treatment according to a predetermined schedule
• Emphasising the importance of regular follow-up and when to request earlier review

It is important to assess the understanding and competencies of the patient before initiating patient education. A study by Hyland et al (2004) found that some patients used the term well controlled asthma in a different sense to clinicians. The researchers suggested that this may be due to an ego-protective mechanism by which patients deny the actuality of asthma. They also found that 1.9% of patients and 21.1% of parents described their asthma or that of their children as very well-controlled but had visited a hospital emergency department or called a physician to the home in the preceding 3 months.

The results of a study by Williams et al (1998) confirmed that patients with poor reading skills do not fully comprehend medical instruction using standard patient education methods.

Different methods can be used to educate patients on their asthma. These include verbal information, written information in the form of pamphlets, videos or audio tapes, asthma classes (individual or in groups) as well as computer programs. The feasibility of these educational methods depends on the resources available for patient education. Urek et al (2004) found that the completion of programs of medical education in asthma classes and individual verbal information significantly increased the general asthma-related knowledge and awareness. In contrast, in group of patients who, besides the standard medical care, received only written information, the asthma-related knowledge remained unchanged.
2.9 PHARMACISTS’ INTERVENTIONS IN ASTHMA MANAGEMENT

Pharmacists in both community, hospital and clinic practice are well placed to provide continued information and reinforcement of key messages to improve compliance with medication and the outcomes of asthma management plans.17

Since 1990 the scope of pharmacy practice globally has changed from a primarily dispensing role to one of a primary health care provider. Pharmacists have extensive knowledge about drug therapy that can assist health care professionals in instructing and educating patients on the correct use of medications. The pharmacist is often the last health care provider the patient will see before going home with their medication and is therefore in a prime position to ensure that the necessary training and education has taken place and to emphasize the important facts that the patient needs to know. Pharmacists may be able to increase medication adherence with counselling and monitoring systems and by facilitating communication with physicians. However, regardless of all of this, it remains uncertain whether pharmacist-patient interactions improve patient outcome12, and in spite of recommendations for teamwork and a multidisciplinary approach in education of asthma patients, medical doctors and nurses still carry out the greatest part of patients’ education.70

Pharmaceutical care is a concept that has evolved from many years of research and practice in the profession of pharmacy. This form of professional practice is not designed to replace the role of the physician or any other health care practitioner but rather to meet a need in the health care system that has arisen because of multiple prescribers for a single patient, the explosion in the number of drug products available on the market, the increased complexity of drug therapy and the significant level of drug-morbidity associated with drug use. Strategies to monitor and improve patient compliance are key components in pharmaceutical care plans which are especially useful in the treatment of chronic conditions such as asthma.5

A number of studies have been published investigating the evolving role of pharmacists and their involvement with community-based asthma patients. A study by Charrois et al (2004)6 suggests that asthma patients are more satisfied with their
pharmacy care if they believed their pharmacist was able to help them manage their asthma. This study was a randomised controlled trial with patients randomised to either an Intervention Group or Control Group. Patients were high-risk asthma patients (defined as having an emergency room visit or hospitalisation in the previous year, or using more than two canisters of short-acting β agonist in the previous 6 months). They were identified through community pharmacies. The primary objective was to determine the effect of an education and referral intervention program initiated by community pharmacists, working with high-risk asthma patients, family physicians and respiratory therapists, on asthma control.

Guided by the philosophy of pharmaceutical care, the aim of pharmacists is to identify and respond to the drug-related needs of people and reduce the risk of drug-related problems. Several studies cited by Chan et al (2004) have documented that inclusion of a pharmacist in a clinic led to reduced drug costs, possibly by minimising drug interactions, hospitalisations and emergency room visits, thereby improving the perception of the quality of health care provided by pharmacists in the health care service.

In a study by Hyland et al (2004), which aimed at assessing treatment needs in patients with asthma, the researchers found that asthma patients may interpret medical terminology differently to clinicians. In this regard, pharmacists can assist patients in understanding the medical terminology used in order for them to follow orders and advice given by health care providers. The study showed some differences between clinicians' and patients or parents' perceptions of treatment. For patients, ‘side effects’ meant long-term effects (10-20 years); for clinicians, it meant occasional problems. Although this study described considerable satisfaction among patients with drug-related asthma information, the study also showed that patients wanted a wider range of information than that normally provided by clinicians. Patient information needs were not only restricted to drug management but also included information about non-drug management and trigger avoidance.

Chan et al (2004) assessed the economic and humanistic outcomes of clinical pharmacist interventions for patients with asthma. Seventy patients who were between 17 and 53 years of age with moderate to severe asthma and who were attending the
outpatient clinic were enrolled in the study. Patients were educated about their disease, pharmacotherapy, self-management as well as inhalation and peak flow meter techniques. Quality of life was assessed using a questionnaire. An asthma general knowledge questionnaire and an asthma diary chart were also used to assess patients’ knowledge about asthma and the improvement in their symptoms. The cost effectiveness was evaluated based on the reduction in total costs or mean cost at each visit. The study found that pharmacist intervention can be a cost-effective addition to the management of patients with moderate to severe asthma at an outpatient clinic. The pharmacist intervention resulted in improved PEFR, improving patient quality of life, and savings in the use of medical resources.

The most important goals in asthma management are improving patients’ everyday functioning, their emotional and social lives and subjective well-being. Therefore, it would be logical to assume that should a demonstrable improvement in quality of life occur, the main objective of pharmaceutical care could be considered to be achieved.9

In a study by Kheir et al (2001)9 that was aimed at assessing the impact of pharmaceutical care specialist asthma services provided by community pharmacists to a sample of patients with asthma, the outcome indicators used were changes in health status and quality of life. Sixty-two adult asthma patients (17 years and older) living in two rural regions of New Zealand, were divided into two groups for phased introduction to the service. The patients acted as their own controls. The results of this study suggest that, with appropriate training and support, pharmacists can help asthma patients achieve greater quality of life.

In a systematic literature review of randomised control studies published in English between 1990 and 2003 by Roughead et al (2005)10, improvements in medication use amongst patients following pharmaceutical care services were demonstrated. Improvements in patient knowledge and adherence were less obvious; however, this may have been because baseline adherence and knowledge rates were high. When viewing the results from this study collectively it becomes apparent that pharmaceutical care is most likely to be beneficial in patient groups whose knowledge and compliance rates are poor.
Weinberger *et al* (2003)\(^{12}\) found that a pharmacist care programme increased patients’ PEFR compared with usual care, but provided little benefit over peak flow monitoring alone. This study was aimed at assessing the effects of a pharmacist care programme for people with asthma and COPD and was a randomised control study conducted at 36 community pharmacies in Indianapolis, USA.

A prospective randomised controlled trial aimed at implementing and assessing a community-based pharmaceutical care programme for patients with asthma was conducted by Cordina *et al* (2001).\(^{11}\) Twenty-two community pharmacies were included in two groups, eleven in the Control Group and eleven in the Intervention Group of pharmacies. A comprehensive asthma education and monitoring programme was implemented. Patients in the Intervention Group of pharmacies received verbal counselling, an educational video, an information leaflet, and subsequent monitoring with reinforcement whilst in the Control Group of pharmacies patients received routine dispensing services. Health-related quality of life of patients who received the pharmacist intervention improved at 12 months. In the same time period, PEFR significantly decreased in control patients compared with intervention patients whereas inhaler technique improved in the intervention group. There were significantly fewer self-reported hospitalisations in intervention patients.

Based on the above, the present study will aim to determine the impact of pharmaceutical care services on the management of adult asthma patients over a three month period at a primary health care clinic.
CHAPTER 3
RESEARCH METHODOLOGY

A randomised control study was conducted at the Kruisfontein Clinic, Humansdorp, Eastern Cape, South Africa. Approval was obtained from both the NMMU Human Ethics Committee (Appendix 1) and the Authorities of the Kruisfontein Clinic (Appendix 2).

3.1 STUDY SETTING

Kruisfontein Clinic is one of two primary health care clinics in Humansdorp and serves the primarily coloured population residing in the surrounding areas of Arcadia, Graslaagte, Vaaldam and Kruisfontein.

3.2 SELECTION OF PATIENTS

Patients, between the ages of 21 and 65, with previously diagnosed asthma (according to their clinic medical records), who were attending the clinic for consultation with clinic staff or collection of medication, were invited to participate in the study. Invitations were made to patients by the researcher who was assisting in the pharmacy with dispensing. Only patients visiting the clinic themselves were invited to participate. Other chronic or acute co-morbidities did not exclude patients from participation in the study.

Patients were invited to participate if their chronic medication indicated that they were asthmatic patients. A standard approach was used each time whereby patients would be informed of the nature of the study and then asked to participate afterwards. Both a written (Appendix 3) and verbal explanation of the study were provided to study participants and those willing to sign a standard letter of informed consent (Appendix 4) were included.
Each patient included in the study (n=120) was given a sequential recruitment number (0001-0120) and allocated to either a Control Group (n=60) or Intervention Group (n=60) by a randomisation process using Random Allocation Software developed by Saghaei. Appointments were set up with individual patients in both groups for an initial assessment interview. Interviews were conducted on Tuesdays, Wednesdays and Thursdays at the clinic. A number of patients invited to participate did not turn up for their first interviews and were then not included in the study.
3.3 PRE-INTERVENTION ASSESSMENTS

The initial assessment interview for both Control and Intervention groups included:

1. Collection of general patient data including age, gender, height, cigarette smoking history, home domestic fuel use, occupation, details of acute and chronic co-morbidities, current and recent (past 6 months) medication history using a purposely designed patient data collection form (Appendix 5).

2. Collection of asthma-related patient data including, onset of asthma, medication and symptom history using a purposely designed patient data collection form.

3. Completion of a Knowledge Attitude and Self-Efficacy Asthma Questionnaire (KASE-AQ) developed by Wigal et al (1993)\(^5\) which assesses patients’ knowledge regarding asthma, their attitudes about their asthma and their self-efficacy regarding their perceived ability to control the disease. This questionnaire developed by Wigal et al is especially useful since it measures all three elements of asthma knowledge, namely their knowledge regarding asthma, their attitudes regarding their asthma (including their willingness to cooperate with their physician in managing asthma) and their self-efficacy regarding their perceived ability to control the disorder.\(^5\) These changes in patient variables are assessed following a particular intervention. Permission to use the questionnaire and scoring information was obtained from Dr JA Winder. The questionnaire was modified slightly to accommodate South African statistics. Illustrations were used to help in the answering of some of the questions (Appendix 6).

4. Completion of the short form of the Asthma Quality of Life Questionnaire AQLQ(S) (Appendix 7) which was obtained from Prof Juniper, in Afrikaans, the patients’ first language.\(^42\) This questionnaire covered the main spheres of human living, which included work place and home assignments, sleeping and relaxation, sport and recreation, family affairs, social contacts, health care, and material status. The short form of this questionnaire, namely the AQLO(S) had also been validated by Juniper et al as having the same measuring properties as the original AQLQ however it was more practical to use since it did not contain the five patient-specific activities which have been proven to change
over time. In the AQLQ(S) a generic list of activities had been given and this problem had therefore been eliminated.

5. An assessment of FEV$_1$, %FEV$_1$, FVC, %FVC, PEFR and %PEFR using a spirometer (Spirobank®) and instruction in the use of this instrument was provided to patients in both groups.

6. Assessment of the patient’s proficiency in the use of an MDI was performed using a method described by Williams et al.$^{57}$ Patients were asked to demonstrate their inhaler technique using their own inhaler or a placebo and this was rated according to a six-step scale to assess their ability to correctly (1) remove the MDI cap and shake the inhaler, (2) exhale slowly prior to inhalation, (3) actuate the MDI at onset of inhalation, (4) inhale at less than maximal rate, (5) hold breath after inhalation for at least 5 seconds, and (6) wait at least 30 seconds between each MDI actuation.

All patient data were documented on a purpose-designed data collection form. (Appendix 5)

Questionnaires were filled in according to patient response when shown the response cards. Some patients requested to fill in their own questionnaires and they were then given the opportunity to do so.
3.4 INTERVENTION

In addition, the therapeutic regimens of the Intervention Group were evaluated against the Standard Treatment Guidelines for the management of asthma in adults at the primary health care level and, where necessary, prescribing recommendations were made. Any suggested prescribing changes were recorded. Patients in this group were also educated on their disease, pharmacotherapy and self-management using purposely designed educational material (Appendix 8). Patient education was on an individual basis at the end of the initial assessment interview. The educational material was used in a printed format and reinforced with verbal information. Patients were also encouraged to ask questions. Since many patients could not read in the study population no written information was given to the patients.

The use of a 500ml plastic bottle as a spacer device (as described by Zar et al\cite{zar62}) which was appropriately modified in order to tightly fit an inhaler mouthpiece was also suggested and provided. Figure 3.1 illustrates how a patient would use the plastic bottle as a spacer.

![Figure 3.1 Modified plastic bottle used as spacer](image)

The Control Group was still attended to by the clinic staff in the usual manner and received their medication from the clinic as previously but did not receive any pharmacist interventions.
3.5 POST-INTERVENTION ASSESSMENT

A follow-up interview was conducted with patients in both study groups three months after the initial assessment interview and again included the following as per the initial assessment:

1. Completion of the AQLQ(S) questionnaire
2. Completion of the KASE-AQ questionnaire
3. Assessment of inhaler technique
4. Spirometry measurements

In the Intervention Group the extent to which prescribing changes were accepted and adopted by prescribers was documented.

After the completion of the study, the pharmaceutical care services provided to the Intervention Group were also made available to those in the Control Group who required them.
3.6 SUMMARY OF THE STUDY DESIGN

The study design is summarised in Figure 3.2 below.

![Study Design Diagram]

- **21-65 years**
- **Moderate to severe asthma**
- **Possible other chronic conditions**
- **Informed consent**
- **Inclusion into study**
- **Allocation to group (n=120)**
- **Control Group (n=60)**
- **Intervention Group (n=60)**
- **Set up appointments**
- **Quality of Life Questionnaire**
- **Completion of asthma knowledge questionnaire**
- **Peak flow assessment**
- **Spirometry assessment**
- **Assessment of inhaler technique**
- **Conducting of second interviews with patients (n=42)**

- **3 MONTHS**
- **Quality of life questionnaire**
- **Peak flow assessment**
- **Spirometry assessment**
- **Assessment of inhaler technique**
- **Filling out of asthma knowledge questionnaire**

- **Provision of asthma education**
- **Changes to medication**
- **Suggest use of home-made spacer device**

**Figure 3.2 Study design**
3.7 ANALYSIS OF RESULTS

Descriptive statistics were used to analyse both the Control and Intervention groups. Changes in PEFR, FEV₁, MDI use and asthma-related quality of life, knowledge, attitudes and self-efficacy scores, over time, both within and between the two groups were analysed. The data were also analysed for outliers. For continuous variables (for example asthma knowledge, attitude and self-efficacy scores) which were normally distributed, the mean change in scores between the Intervention and Control groups was assessed by the unpaired Student’s t-test. Paired Student’s t-tests were used to assess the mean changes from baseline for each outcome within the groups. Analysis of covariance (ANCOVA) was used to adjust for those characteristics that were unequally distributed at baseline, such as age, and therefore could potentially confound the association between the Intervention Group and outcome variables. In all statistical tests, p-values of <0.05 were considered to be statistically significant. Cohen’s $d$ and the adjusted means were also determined for all statistically significant results. Cohen's $d$ is the appropriate effect size measure used in the context of a t-test on means. Cohen in 1992 determined that a $d$ value between 0.2 and 0.5 is indicative of a small effect, 0.5 to 0.8 is indicative of a medium effect and 0.8 and larger is indicative of a large effect size.⁷³ All data analyses were performed using statistical software (Statistica version 7.0®). All statistical analyses were completed in consultation with a statistician.
The total number of patients entered into the study was 120. Patients were allocated to either the Control or Intervention group. All patients completed the baseline questionnaires. Of those randomly assigned to the Intervention Group (n=60), 40 (66.67%) returned for the second interviews and completed the follow-up questionnaires and assessments. Of those assigned to the Control Group (n=60), 42 (70.0%) returned for the second interviews and completed the follow-up questionnaires and assessments. Patients were given appointment cards and were given verbal instructions for the follow-up visit. Lists with names and appointment times were posted on the notice board in the clinic waiting room and patients who provided telephone numbers were contacted telephonically to remind them of their next appointment. Patients were also reminded of their follow-up visit when they came to collect chronic medication from the nursing sister at the clinic. The reasons for the poor follow-up return of patients may have been attributed to transport difficulties, unfavourable weather conditions in which patients were not able to walk to the clinic, as well as the low level of literacy which prevented patients from reading the appointment card given to them with the date and time of their next appointment.
4.2 BASELINE CHARACTERISTICS

The following table provides a summary of the baseline characteristics of both the Control and Intervention groups:

Table 4.1 Baseline characteristics of participants included in the study

<table>
<thead>
<tr>
<th></th>
<th>Control Group (n=60)</th>
<th>Intervention Group (n=60)</th>
<th>t-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No of males (%)</td>
<td>21 (35)</td>
<td>21 (35)</td>
<td>0.0</td>
<td>1.0</td>
</tr>
<tr>
<td>No of females (%)</td>
<td>39 (65)</td>
<td>39 (65)</td>
<td>0.0</td>
<td>1.0</td>
</tr>
<tr>
<td>No of smokers (%)</td>
<td>24 (40)</td>
<td>29 (48)</td>
<td>0.9</td>
<td>0.4</td>
</tr>
<tr>
<td>No of home fuel users (%)</td>
<td>14 (23)</td>
<td>13 (22)</td>
<td>-0.2</td>
<td>0.8</td>
</tr>
<tr>
<td>Mean age in years (SD)</td>
<td>50.38 (9.18)</td>
<td>46.82 (9.25)</td>
<td>-2.1</td>
<td>0.04</td>
</tr>
<tr>
<td>Mean height in m (SD)</td>
<td>1.62 (0.10)</td>
<td>1.61 (0.10)</td>
<td>-0.6</td>
<td>0.5</td>
</tr>
<tr>
<td>Mean weight in kg (SD)</td>
<td>65.39 (20.69)</td>
<td>69.82 (18.30)</td>
<td>1.2</td>
<td>0.2</td>
</tr>
<tr>
<td>QOL questionnaire (SD):</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Activity limitation (best=7)</td>
<td>2.7 (0.69)</td>
<td>2.54 (0.63)</td>
<td>-1.36</td>
<td>0.18</td>
</tr>
<tr>
<td>Symptoms (best=7)</td>
<td>2.61 (0.87)</td>
<td>2.20 (0.95)</td>
<td>-2.46</td>
<td>0.02</td>
</tr>
<tr>
<td>Emotional function (best=7)</td>
<td>2.28 (1.05)</td>
<td>1.87 (0.85)</td>
<td>-2.39</td>
<td>0.02</td>
</tr>
<tr>
<td>Environmental stimuli (best=7)</td>
<td>1.65 (0.71)</td>
<td>1.52 (0.70)</td>
<td>-1.0</td>
<td>0.32</td>
</tr>
<tr>
<td>Total (max=224)</td>
<td>79.12 (20.94)</td>
<td>69.80 (22.67)</td>
<td>-2.34</td>
<td>0.02</td>
</tr>
<tr>
<td>Average (max=7)</td>
<td>2.47 (0.65)</td>
<td>2.18 (0.71)</td>
<td>-2.34</td>
<td>0.21</td>
</tr>
<tr>
<td>KASE-AQ (SD):</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knowledge (max=20)</td>
<td>12.32 (4.23)</td>
<td>10.78 (2.92)</td>
<td>-2.31</td>
<td>0.23</td>
</tr>
<tr>
<td>Attitude (max=100)</td>
<td>77.82 (8.64)</td>
<td>73.98 (5.56)</td>
<td>-2.89</td>
<td>0.005</td>
</tr>
<tr>
<td>Self-efficacy (max=100)</td>
<td>71.20 (9.13)</td>
<td>67.55 (6.51)</td>
<td>-2.52</td>
<td>0.01</td>
</tr>
<tr>
<td>Total (max=220)</td>
<td>161.33 (16.92)</td>
<td>152.32 (10.03)</td>
<td>-3.55</td>
<td>0.0006</td>
</tr>
<tr>
<td>%PEFR mean (SD)</td>
<td>27.67 (15.19)</td>
<td>28.15 (17.57)</td>
<td>0.16</td>
<td>0.87</td>
</tr>
<tr>
<td>%FVC mean (SD)</td>
<td>46.92 (16.81)</td>
<td>44.80 (20.62)</td>
<td>-0.62</td>
<td>0.54</td>
</tr>
<tr>
<td>%FEV1 mean (SD)</td>
<td>43.27 (17.44)</td>
<td>43.33 (21.15)</td>
<td>0.02</td>
<td>0.98</td>
</tr>
<tr>
<td>Inhaler technique (max=7)</td>
<td>2.75 (1.23)</td>
<td>3.12 (1.26)</td>
<td>1.61</td>
<td>0.11</td>
</tr>
</tbody>
</table>

* Indicates baseline characteristics that showed a significant difference (p<0.05) between Control and Intervention groups

Baseline data were analysed for outliers and none were found.
4.1.1 Demographics of the sample population

4.1.1.1 Gender

In both the Control and Intervention groups 35.0% of patients were male and 65.0% were female (p=1.0). The fact that more women were included in the study can possibly be attributed to the fact that men are normally at work during the day when the clinic is open and women therefore attend the clinic to collect chronic medication for both themselves and their partners. Women may also tend to show a greater interest in their condition when compared with men as was shown in a study by Alexandre et al (2002) which included 248 patients and was aimed at predicting compliance with short-term treatment in patients with back pain. One of the findings was that women showed more interest in their disease and were more likely to be compliant with short-term medication when compared with men.

4.1.1.2 Smoking

Of all the patients allocated to the Control Group 40.0% were smokers compared with 48.0% of patients in the Intervention Group (p=0.4). These percentages relate closely to the national statistics for the incidence of smoking in adults according to the South African Demographics and Health Survey of 1998, which showed that 42% of males and 11% of women are smokers. The high incidence of smoking in asthmatic patients is of concern and can possibly be related, in part, to a lack of education. The patients included in this study were encouraged to stop smoking and educated on the effects of smoking on the lungs and general health. The role of tobacco smoking in respiratory disease in South Africa was confirmed in the South African Demographics and Health Survey of 1998, by the finding that at all ages, men and women, who had smoked have a higher prevalence of chronic bronchitis and also of episodic airflow limitation and abnormal PEFR, than people who had never smoked before.
4.1.1.3  **Home fuel use**

Of all the patients allocated to the Control Group 23.0% made use of home fuel such as paraffin compared with 22.0% in the Intervention Group. These percentages are slightly lower than the national average of 33.3% according to the South African Demographics and Health Survey of 1998. According to this survey there was a moderately strong association in males between exposure to smoke, dust, fumes or strong smells and both airflow limitation and chronic bronchitis, and a little less so for PEFR abnormality.

4.1.1.4  **Age of participants**

The mean age for the whole study population group was 48.6 years with a standard deviation of 9.4 years (range 29-65). The mean age for the Control Group was 50.4 years (SD 9.2; range 29-65) and for the Intervention Group the mean age was 46.8 years (SD 9.3; range 29-65).

The following histogram illustrates the age distribution of patients in the Control and Intervention groups:

![Figure 4.1 Age distribution in the Control and Intervention groups (n=120)](image-url)
In the Control Group 26.67% of the patients were below the age of 45 years whilst 48.33% of patients in the Intervention Group were below the age of 45 years.

Age showed the only significant difference in the demographic composition of the Control and Intervention groups. There was a mean difference of fourteen years (Student’s t-test; p=0.04). Age was added as a variable in the post-intervention statistical modelling and analysis and it was found that it did not have a significant effect on the results.

4.1.1.6 Height and weight

The mean height of all patients in the study population was 1.61m with a standard deviation of 0.10m and the mean weight was 67.60kg with a standard deviation of 19.60kg. The mean height was 1.62m with a standard deviation of 0.10m in the Control Group and 1.61m with a standard deviation of 0.10m in the Intervention Group. The mean weight was 65.39kg with a standard deviation of 20.69kg in the Control Group and 69.82kg with a standard deviation of 18.30kg in the Intervention Group.

There was no significant difference in either height or weight between the Control and Intervention groups (p=0.5 and 0.2 respectively).

The significance of measuring the height and weight of the patients is that both height and weight are used in the determination of spirometric values. Normal spirometric values will increase proportionally as height and weight increases.29

4.1.2 Asthma severity

The asthma severity of the patients was assessed based on the frequency of night-time symptoms, the frequency of daytime symptoms and the %PEFR. Assessment was performed on the basis of a rating scale. Due to the fact that the %PEFR values recorded for the patients were very low, an accurate assessment of asthma severity could not be performed and therefore this was not included in the study. One possible way of assessing asthma severity could be to assess the medication needed to control
the asthma and to then categorise the patient’s asthma severity according to the medication needed. Asthma severity assessments should consider not only patients’ physiologic and symptom measures but also recent medication and health care utilization.28

The low spirometry values obtained during the study were discussed in person with the clinic doctor and he agreed that he experienced the same problems when measuring lung function in asthmatic patients.

The %PEFR readings may have been low because patients were hesitant to use the spirometer correctly. Many of them receive disability grants from the government based on the fact that they are unable to work due to the severity of their asthma. Patients are absolutely reliant on these disability grants for their everyday needs and are very hesitant to show an improvement in asthma symptoms as they feel this might cause them to lose their grants.†

4.1.3 Age of onset of asthma

The age of onset of asthma was recorded but since the patient files were not very reliable or current, the quality of the data collected was questionable. The current system of keeping patient records was only introduced in 1995 and any information regarding patient medication and symptoms before 1995 is therefore based solely on the individual patient’s recall of events.

† These issues were discussed in personal communication with the doctor at the clinic.
4.1.4  Co-morbid disease states in the Control and Intervention groups

Patient files were assessed for co-morbid disease states. The following graph indicates the co-morbid disease states for the Control and Intervention groups respectively:

![Graph showing co-morbid disease states](image)

**Figure 4.2  Co-morbid disease states in the Control and Intervention groups (n=120)**

The most prevalent co-morbid disease state was hypertension in both the Control and Intervention groups. A total of 23 patients in the Control Group and 25 patients in the Intervention Group suffered from co-morbid hypertension. This is of particular importance when looking at potential drug interactions and contraindications with asthma and antihypertensive medication. Theophylline is relatively contraindicated in hypertension, epilepsy and congestive cardiac failure. It also has positive inotropic and chronotropic effects on the heart and can therefore exacerbate hypertension. The use of theophylline in hypertensive patients is of concern in the study population since pre-intervention 91.67% of patients included in the study were prescribed theophylline and 40.00% of these patients suffered from co-morbid hypertension. Concomitant use of salbutamol and digoxin or diuretics increases the risk of cardiac arrhythmias. Salbutamol should also be used with caution in hypertensive patients due to the risk of tachycardia and palpitations.

A total of 58 patients did not have any co-morbid diseases (28 and 30 in the Control and Intervention groups respectively). Since patients without co-morbidities will tend to take fewer drugs on a daily basis they are more likely to have less drug interactions.
and side effects. An absence of co-morbidities should also improve the adherence of patients since previous studies have suggested that poor adherence was positively associated with co-morbidity. Studies by Alexandre et al.\textsuperscript{74} and Bender\textsuperscript{75} found that patients were more likely to be non-adherent with medication regimens when one or more co-morbidities were present.

4.1.5 Other medication prescribed in the Control and Intervention Groups

Patient files of the Control and Intervention groups were assessed for other medications prescribed for the patient. A summary of this information is presented in Figure 4.3.
Figure 4.3 Other medication prescribed in the Control and Intervention groups (n=120)
The drugs most often co-prescribed with the asthma medication were perindopril and hydrochlorothiazide. A total of 16 patients in the Control Group and 17 patients in the Intervention Group were prescribed perindopril. In the Control Group 18 patients were using hydrochlorothiazide compared with 10 patients in the Intervention Group. The concomitant use of hydrochlorothiazide and corticosteroids increases the risk of hypokalemia and is therefore of concern in asthmatic patients using chronic oral corticosteroids. During the intervention the patients’ other medication was taken into account before changes to medication were suggested.

β blockers such as propranolol, used in the treatment of hypertension are contraindicated in asthma due to the β-receptor antagonism which causes bronchoconstriction. The cardioselective β blockers, for example atenolol, are also contraindicated in asthma due to the risk of bronchoconstriction. Two patients in the Intervention Group were taking atenolol. After consultation with the doctor it was decided that the use of atenolol in both these patients should be stopped. Nifedipine 30mg once daily was started in both these patients to control hypertension.

Due to the risk of tachycardia, the use of inhaled salbutamol in a patient with congestive cardiac failure was also stopped after recommendations to the clinic doctor were made. The doctor decided that the difficulty in breathing that this patient was experiencing was due to congestive cardiac failure and not due to asthma and asthma medication was therefore not necessary.

The prescribed medication assessed did not include any tuberculosis, anti-depressant or anti-psychotic medication or anti-retroviral therapy since these are not indicated on the patient files and are kept in separate files by the sister dealing with tuberculosis patients, the psychiatric mobile clinic and the sister dealing with HIV patients respectively.

Tuberculosis medication can have a profound effect on asthma medication, especially on theophylline. Both rifampicin and isoniazid can significantly affect theophylline plasma levels. Rifampicin is a potent liver enzyme inducer and can therefore reduce the efficacy of theophylline. Isoniazid, on the other hand, inhibits cytochrome P450 enzymes and concomitant use of theophylline and isoniazid can therefore lead to
increased plasma levels of theophylline, putting the patient at risk of theophylline toxicity.\textsuperscript{48} The same holds true for some of the psychiatric medicines such as fluoxetine as well as the antiretroviral agents, especially the protease inhibitors which are reserved as second-line therapy in anti-retroviral therapy (ART), all of which are also not recorded on the patient files. It would be ideal to record all these medication on the patients’ files since the possibility of drug interactions would be minimised if the prescriber and pharmacist can assess what other medication patients are taking before suggesting or making changes to medication regimens.

4.1.6 Average number of items per prescription

The average number of items on each patient’s prescription was assessed and the following graph indicates the average number of items per prescription for the Control and Intervention groups respectively. This includes all medication reported as chronic medicine in the patient file except for tuberculosis medication, psychiatric medication and anti-retroviral medication.

![Average number of items prescribed in the Control and Intervention groups (n=120)](image)

The average number of items per prescription in the Control and Intervention groups was 3.4. This is more than the national average of 2.3 items per prescription published by the South African Department of Health in 1998.\textsuperscript{76} A total of two items on a prescription for an asthmatic patient at this clinic was in most cases only asthma medication, theophylline SR 250mg tablets and a salbutamol inhaler being the most
common combination. The compliance of a patient with fewer items on a chronic prescription is better than that of a patient who has to take, for example, ten drugs every day. The possibility of drug interactions and side effects are of concern especially with the patient in the Control Group who had ten items on his prescription. This particular patient was an epileptic with co-morbid hypertension, asthma and congestive cardiac failure. He was prescribed phenytoin sodium 300mg nocte, phenobarbitone 60mg nocte, spiranolactone 50mg twice daily, furosemide 80mg twice daily, theophylline SR 250mg twice daily, inhaled salbutamol as needed, inhaled beclomethasone 100µg twice daily, one vitamin B complex tablet daily, sublingual isosorbide dinitrate 5mg as needed and perindopril 2mg mane. Also of concern is the fact that tuberculosis medication, antiretrovirals and psychiatric medication are not part of this average number of items and can therefore further complicate issues of side-effects, contraindications, drug interactions and compliance if were to be prescribed any of these.

4.1.7 QUALITY OF LIFE QUESTIONNAIRE BASELINE VALUES

As part of the baseline interview the short form (32 question) Quality of Life (QOL) Questionnaire by Juniper et al (1999) was completed for all patients included in the study. Some patients indicated that they would prefer to fill in their own questionnaires and they were then given the opportunity to do so. Juniper et al (1999) noted that there was very little difference in overall scores when questionnaires are completed by patients as compared to when questionnaires are administered by an interviewer, although it is recommended to stick to one method when the patient is being followed over time. There is also minimal risk of bias when patients consistently have the questionnaire administered by an interviewer while the rest of the patients complete it on their own.

4.1.7.1 Total QOL scores

The QOL questionnaire is composed of four subscales namely symptoms, activity limitation, emotional function and environmental stimuli. The total for the QOL questionnaire is calculated by adding up the scores for the 32 questions. The maximum score for the total quality of life questionnaire is 224.
The following histogram indicates the distribution of the total scores for the QOL questionnaire:

![Histogram](image)

**Figure 4.5** Total scores for the QOL questionnaire in the Control and Intervention groups (n=120)

In the Control Group a mean total QOL score of 79.12 with a standard deviation of 20.94 was reported. The mean reported in the Intervention Group was 69.78 with a standard deviation of 22.67. This indicates that the patients’ QOL was dramatically reduced because of asthma. A p-value of 0.02 was calculated, indicating a significant difference in baseline total QOL score in the Control and Intervention groups. The highest score for the total QOL in the Control Group was 150 and the lowest score was 46. The highest score for the total QOL in the Intervention Group was 141 and the lowest 41.

The Control Group showed higher baseline values when compared with the Intervention Group for all subscales of the QOL questionnaire.
4.1.7.2 Average QOL scores

The average for the QOL questionnaire is calculated by dividing the total for the QOL questionnaire by the number of questions that make up the questionnaire which is 32. The following histogram indicates the average scores for the QOL questionnaire in the Control and Intervention groups:

![Histogram showing QOL scores](image)

**Figure 4.6 Average scores for the QOL questionnaire in the Control and Intervention groups (n=120)**

The average is used to assess the impact of an intervention on asthma-related QOL. A change in score of 0.5 on the seven point scale is considered clinically meaningful and is regarded as the minimal important difference (MID).\(^{42}\)

In the Control Group the mean average for the QOL questionnaire was 2.47 with a standard deviation of 0.65. The mean average for the QOL questionnaire in the Intervention Group was 2.18 with a standard deviation of 0.71. This is on the severe end of the seven point scale since an average score of 7.0 indicates no impairments due to asthma and 1.0 indicates severe impairment of quality of life due to asthma.

A study by Juniper et al (2005)\(^{77}\) showed a baseline average QOL score of 4.95 (p=0.063) in patients older than eighteen years. This study was aimed at comparing
the measurement properties of the Asthma Quality of Life Questionnaire (AQLQ) questionnaire in patients 12–17 years and patients 18 years and older. A total of 2433 patients (12–75 years) with current asthma and with data that could be evaluated both at randomisation and at the end of treatment were included.

A study by Kheir et al (2001)\textsuperscript{9} reported a baseline mean average QOL score of 4.8 with a standard deviation of 0.9. Sixty-two adult asthma patients (17 years and older) living in two rural regions of New Zealand, were segregated into two groups for phased introduction to the service. The patients acted as their own controls before they received the pharmacists’ service which involved individualised education on drug therapy and usage of medication, demonstration of inhaler technique and the ways to identify and avoid asthma triggers. If necessary, the pharmacist referred the patient to a general practitioner for specific assessment and management. The study was aimed at assessing the impact of a pharmaceutical care specialist asthma service provided by community pharmacists, the outcome indicators being changes in health status and QOL.

The average QOL score in the present study population was therefore low in comparison with the other two studies. There was no significant difference in baseline average QOL scores in the Control and Intervention groups with a p-value of 0.21.

4.1.7.3 Activity limitation scores

The QOL questionnaire included 11 generic questions measuring the degree of activity limitation due to asthma in the two weeks prior to the interview. The types of questions asked to assess activity limitation included questions on climbing or running up stairs, sport, social activities, work related activities as well as sleeping.
The following histogram illustrates the distribution of scores in the Control and Intervention groups for the activity limitation subscale of the QOL questionnaire:

In the Control Group the mean for the activity limitation subscale of the QOL questionnaire was 2.71 with a standard deviation of 0.69. The mean for the same subscale in the Intervention Group was 2.54 with a standard deviation of 0.63. Again, this is on the severe end of the seven point scale. No significant difference in baseline mean activity limitation score was recorded as a p-value of 0.18 was calculated even though the Control Group scored higher than the Intervention Group for baseline mean activity limitation score.

The study by Juniper et al (2005)\textsuperscript{77} produced a baseline score of 5.09 (p=0.002) for the activity limitation subscale of the QOL questionnaire. Kheir et al (2001)\textsuperscript{9} reported a baseline mean activity limitations score of 4.8 with a standard deviation of 0.8. Patients in the present study therefore showed a reduced baseline QOL due to asthma when considering activity limitation.
4.1.7.4 Symptoms scores

For the symptoms subscale of the QOL questionnaire, a total of 12 questions tested the degree of quality of life impairment due to symptoms of asthma in the two weeks prior to the interview. Questions asked to assess symptoms regarding asthma included questions on a tight chest, coughing, shortness of breath, wheezing and day- or nighttime symptoms. The following figure illustrates the distribution of scores for the symptoms subscale of the QOL questionnaire in the Control and Intervention groups:

![Bar chart showing distribution of symptoms scores in Control and Intervention groups](image)

**Figure 4.8 Symptoms scores for the QOL questionnaire in the Control and Intervention groups (n=120)**

The mean for the symptoms subscale of the QOL questionnaire in the Control Group was 2.61 with a standard deviation of 0.87. In the Intervention Group the mean for the symptoms subscale was 2.20 with a standard deviation of 0.95. Both of these scores are on the severe end of the seven point scale indicating severe impairment of QOL due to asthma symptoms.

There was a significant difference in baseline mean symptoms score between the Control and Intervention groups (p=0.02) with the Control Group showing higher baseline values.
The study by Juniper et al (2005)\textsuperscript{77} showed a 4.87 baseline score for the symptoms subscale (p=0.40). Kheir et al (2001)\textsuperscript{9} reported a baseline mean symptoms score of 4.5 with a standard deviation of 1.1. Again, the participants in this study scored considerably lower than their counterparts in the 2005 study by Juniper et al and the study by Kheir et al. Patients included in the present study therefore showed a significant limitation on QOL specifically regarding asthma symptoms.

\subsection*{4.1.7.5 Emotional function scores}

The following histogram illustrates the distribution of scores in the Control and Intervention groups for the emotional function subscale of the QOL questionnaire which was measured by a total of 5 questions. Some of the issues addressed by these included patient frustration regarding asthma, concern over the need for medication and the concern of being out of breath.

\begin{figure}[h]
\centering
\includegraphics[width=0.8\textwidth]{fig49.png}
\caption{Emotional function scores for the QOL questionnaire in the Control and Intervention groups (n=120)}
\end{figure}

In the Control Group the mean for the emotional function subscale was 2.28 with a standard deviation of 1.04. The mean for this subscale in the Intervention Group was 1.87 with a standard deviation of 0.85. Both these scores are on the severe end of the seven point scale, again indicating a limitation on QOL due to emotional function regarding asthma.
A p-value of 0.02 (Student’s t-test) was calculated indicating a significant difference in baseline mean emotional function score in the Control and Intervention groups. Patients in the Control Group had a better score for baseline emotional function compared with their Intervention Group counterparts.

Juniper et al (2005)\textsuperscript{77} found the baseline emotional function score to be 5.06 (p=0.68) in their study population of patients older than eighteen years. A baseline mean emotional function score of 4.7 with a standard deviation of 1.4 was reported in the Kheir et al (2001)\textsuperscript{9} study. The participants included in the present study therefore scored lower than the patients included in the other two studies mentioned. Patients included in the sample population for the present study therefore had a diminished QOL due to emotional function regarding their asthma.

4.1.7.6 Environmental stimuli scores

In the environmental stimuli subscale of the QOL questionnaire, questions were asked to determine the impact of environmental stimuli such as exposure to dust, cigarette smoke, strong smells, perfume and the impact of weather changes on the patient’s asthma.
The following histogram indicates the distribution of scores for environmental stimuli:

![Histogram of environmental stimuli scores](image)

**Figure 4.10  Environmental stimuli scores for the QOL questionnaire in the Control and Intervention groups (n=120)**

A total of 4 questions measured the impact of environmental stimuli on asthma-related quality of life in the two weeks prior to the interview. In the Control Group the mean for the environmental stimuli subscale of the QOL questionnaire was 1.65 with a standard deviation of 0.71. The mean for the same subscale of the QOL questionnaire in the Intervention Group was 1.52 with a standard deviation of 0.71. Again, these scores are at the severe end of the seven point scale which would indicate a significantly negative impact on asthma-related quality of life due to environmental stimuli.

No significant difference in baseline environmental stimuli score in the Control and Intervention groups was recorded (p=0.32) even though the Control Group apparently displayed higher baseline scores.

In a similar study by Juniper *et al* (2005) a baseline score of 4.64 (p=0.028) was reported for this subscale of the QOL. Kheir *et al* (2001) reported a baseline environmental stimuli score of 5.1 with a standard deviation of 0.9. Patients in the
present study therefore had a marked limitation on their QOL due to environmental stimuli affecting asthma.

4.1.9 KNOWLEDGE, ATTITUDE AND SELF-EFFICACY SCORES AT BASELINE

The KASE-AQ consisted of sixty questions and tested a patient’s knowledge, attitude and self-efficacy regarding asthma management. At the first visit a KASE-AQ was completed for each patient in the sample group. Patients who preferred to fill in their own questionnaires were again given the opportunity to do so. No evidence was found that this would have any impact on the results of this study in comparison with the results of the questionnaires that were filled in on behalf of the patients.

4.1.9.1 Total KASE-AQ scores

The maximum score that can be achieved with the KASE-AQ is 220. The following histogram illustrates the distribution of scores for patients in the Control and Intervention groups for total KASE-AQ:

![Histogram showing distribution of total KASE-AQ scores](image)

**Figure 4.11** Total KASE-AQ scores in the Control and Intervention groups (n=120)
In the Control Group the mean score for the total KASE-AQ was 161.33 with a standard deviation of 16.92. The mean in the Intervention Group was 152.32 with a standard deviation of 10.03. The baseline score in the Control Group was therefore significantly higher than that of the Intervention Group (p=0.0006).

Overall the knowledge, attitudes and self-efficacy of patients included in the study were low when compared with a study by Put et al (2002). This study was aimed at evaluating an individualised asthma programme directed at behavioural change in asthmatic subjects who reported complaints and impairment, despite adequate medical treatment. Mild-to-moderate asthma patients (n=23) were randomly assigned to an Intervention or Control Group. The Knowledge, Attitude and Self-Efficacy Asthma Questionnaire was one of the outcome measurements used. Both groups were evaluated at three consecutive times, each separated by 3 months; the programme was delivered between the first two evaluations.

Details of the present study are discussed under the subscale headings for the KASE-AQ.

4.1.8.2 Knowledge subscale of KASE-AQ scores

The knowledge subscale of the KASE-AQ consisted of twenty multiple choice questions each with one correct answer. A maximum of 20 could be scored for the knowledge subscale of the KASE-AQ. The total is measured by calculating the number of correct answers given. Asthma knowledge was assessed by asking questions which covered topics such as asthma symptoms, components of the respiratory system, medication side-effects and management of an acute asthma attack.
The following histogram illustrates the distribution of scores for the knowledge subscale of the KASE-AQ in the Control and Intervention groups:

![Histogram showing distribution of scores for knowledge subscale](image)

**Figure 4.12** Knowledge subscale scores for KASE-AQ in the Control and Intervention groups (n=120)

The mean score for the knowledge subscale of the KASE-AQ in the Control Group was 12.32 with a standard deviation of 4.23. In the Intervention Group the mean for the same subscale was 10.78 with a standard deviation of 2.92. Since the baseline values for the knowledge subscale of KASE-AQ had no significant effect on post-intervention values, analysis of variance (ANOVA) was used in statistically analysing the data instead of analysis of covariance (ANCOVA).

As with most of the other scores for the QOL and KASE questionnaires, patients in the Control Group scored higher at baseline compared with patients in the Intervention Group (p=0.02).

The similar study by Put *et al.* (2002) showed scores of 11.8 with a standard deviation of 2.6 in the Intervention Group and 11.0 with a standard deviation of 2.5 in the Control Group for the knowledge subscale of the KASE-AQ. The scores obtained in the present study for the knowledge subscale at baseline are therefore similar to those for the study by Put *et al.* Overall, the pre-intervention scores for knowledge
were acceptable, however it is important to remember that improving a patient’s knowledge of asthma can improve the management of asthma.  

4.3.8.3 Attitude subscale of KASE-AQ scores

The attitude subscale contained 20 questions. Each of these 20 questions was answered on a five point Likert-type scale, from “True” to “False”. Since these questions dealt with the patient’s attitude towards his or her asthma, there were no correct or incorrect responses. However, the higher the score, the more positive the individual’s attitude toward his or her asthma and the more willing and eager the individual was to manage the disorder by working in cooperation with the physician. The lower the score, the more pessimistic and uncooperative the individual’s attitude was towards his or her condition. A maximum of 100 could be scored for the attitude subscale of the KASE-AQ.

The assessment of attitudes about asthma included asking patients about willingness to learn about asthma, the extent to which they trust in their own ability to control the disease and their willingness to cooperate with the doctor in managing the condition.
The distribution of scores in the Control and Intervention groups for the attitude subscale of the KASE-AQ is illustrated in Figure 4.13.

![Figure 4.13 Attitude subscale scores for KASE-AQ in the Control and Intervention groups (n=120)](image)

In the Control Group the mean for the attitude subscale of the KASE-AQ was 77.82 with a standard deviation of 8.64. The mean for the attitude subscale in the Intervention Group was 73.98 with a standard deviation of 5.56. Again, patients in the Control Group scored significantly higher at baseline compared with patients in the Intervention Group for the attitude subscale (p=0.005). ANOVA was used in statistically analysing the data instead of ANCOVA since the baseline values for the attitude subscale of KASE-AQ had no significant effect on post-intervention values.

In the study by Put et al (2002) mean attitude scores of 77.0 with a standard deviation of 4.0 and 76.0 with a standard deviation of 5.0 (p=<0.0001) were reported at baseline in the Intervention and Control groups respectively. Overall, the attitude of the patients included in the present study scored relatively highly on the KASE-AQ and the scores are similar to the scores obtained in the Put et al study.
4.3.8.4 Self-efficacy subscale of KASE-AQ scores

The self-efficacy subscale also contained 20 questions. Each of these 20 questions was also answered on a five point Likert-type scale, from “True” to “False”. Again, these questions dealt with the patient’s self-efficacy regarding his or her asthma and therefore there were no correct or incorrect responses. Questions included to assess self-efficacy covered aspects on the patient’s confidence in taking the necessary steps to avoid or manage an asthma attack, recognising changes in the lungs and confidence in solving problems relating to asthma. The higher the score, the more confident the individual was in his or her ability to manage and control the asthma. A maximum of 100 could be scored for the self-efficacy subscale of the KASE-AQ.

The following histogram illustrates the distribution of scores in the Control and Intervention groups for the self-efficacy subscale of the KASE-AQ:

![Histogram showing distribution of self-efficacy scores](image)

Figure 4.14 Self-efficacy subscale scores for KASE-AQ in the Control and Intervention groups (n=120)

In the Control Group the mean score for the self-efficacy subscale of the KASE-AQ was 71.20 with a standard deviation of 9.13. The mean score for the same subscale in the Intervention Group was 67.55 with a standard deviation of 6.51.
Patients in the Control Group displayed significantly higher baseline scores \((p=0.01)\). The baseline scores for the self-efficacy subscale were lower than the baseline attitude subscale scores for the Control and Intervention Groups.

The study by Put et al (2002)\(^7\) showed mean baseline scores for the self-efficacy subscale of 71.0 with a standard deviation of 8.0 and 67.0 with a standard deviation of 9.0 \((p=<0.0001)\) in the Intervention and Control groups respectively. The baseline self-efficacy scores for the present study were therefore almost exactly matched with the scores obtained by Put et al. Overall the baseline self-efficacy scores were acceptable.

### 4.3.9 SPIROMETRY MEASUREMENTS

Baseline \(\text{FEV}_1\), \(\%\text{FEV}_1\), \(\text{FVC}\), \(\%\text{FVC}\), \(\text{PEFR}\) and \(\%\text{PEFR}\) values were measured using a spirometer (Spirobank\(^8\)). Measurements were taken after explaining to the patient the correct way of using the spirometer.

Extensive clinical studies have concluded that only two measurements are needed in spirometry: \(\text{FVC}\) (a volume test) and \(\text{FEV}_1\) (a flow test). \(\text{FVC}\) is the amount of air that can be blown out of fully inflated lungs, and \(\text{FEV}_1\), is the rate of expiratory air flow. Spirometers require precision within 3\% before displaying \(\text{FVC}\) and \(\text{FEV}_1\) values, so that falsely low results are avoided or at least mitigated.\(^2\)

A peak flow meter is a less expensive way of measuring lung function and can be used in the outpatient management of asthma because it is easier to use and more freely available than spirometers.\(^3\) \(\text{PEFR}\) is less sensitive and more variable and is strongly dependent on how hard the patient tries. It is also dependent on age, sex and body size. The most common method of relating \(\text{PEFR}\) to environmental and other variables of interest is to express \(\text{PEFR}\) as a percentage of a predicted or reference value drawn from a study of a population suitable for this purpose.\(^3\)
4.1.9.1 %PEFR values

The following histogram illustrates the distribution of %PEFR values in the Control and Intervention groups:

![Histogram](image)

**Figure 4.15 Baseline %PEFR values in the Control and Intervention groups (n=120)**

The mean %PEFR in the Control Group was 27.67% with a standard deviation of 15.19%. In the Intervention Group patients displayed a mean %PEFR of 28.15% with a standard deviation of 17.57%. A p-value of 0.87 indicated no significant difference in baseline %PEFR in the Control and Intervention groups. On average the readings for %PEFR were low.

Ngamvitroj et al (2005) conducted a study aimed at determining the effects of asthma self-efficacy, perceived satisfaction with social support and asthma knowledge on adherence to PEFR self-monitoring behaviour. The researchers also examined whether adherence to PEFR self-monitoring mediates the effects of psychosocial/cognitive factors on lung function and asthma symptoms in adults with asthma. Sixty-eight participants completed standardised questionnaires three times: at baseline, 1 month, and 3 months and kept the records of PEFR self-monitoring behaviours twice a day. A mean baseline morning %PEFR value of 66.47% with a standard deviation of 24.12% was recorded.
The %PEFR values obtained for patients in the present study were therefore very low compared with the results of the study by Ngamvitroj et al but could be attributed to the fact that patients were very wary of using the spirometer correctly as suggested in section 4.1.2. The patient’s severity of asthma could also not be accurately determined since this is also reliant on the %PEFR which could not be measured accurately.

4.1.9.2 %FVC values

The following histogram illustrates the %FVC baseline values for the Control and Intervention groups:

![Figure 4.16 Baseline %FVC values in the Control and Intervention groups (n=120)](image)

In the Control Group the mean %FVC at baseline was 46.92% with a standard deviation of 16.81%. The mean in the Intervention Group at baseline was 44.80% with a standard deviation of 20.62%. A p-value of 0.54 was calculated, indicating no significant difference in baseline %FVC in the Control and Intervention groups. Again, the %FVC values were low.

Kauppinen et al (1999) conducted a randomised control study involving patient education on asthma self-management. An individual education session was
conducted at baseline with every patient included in the study. At three, six and nine months, thirty minute individual educational sessions were held with patients in the Intervention Group. The study included 162 patients and follow-up values were measured at 12 months. A baseline mean %FVC of 94.7% was recorded in the Intervention Group. Again, the values obtained for mean baseline %FVC in the present study were therefore low compared with the study by Kauppinen et al.

4.1.9.3 %FEV₁ values

Figure 4.17 illustrates the %FEV₁ baseline values for the Control and Intervention groups:

![Figure 4.17 Baseline %FEV₁ values in the Control and Intervention groups (n=120)](chart)

The mean %FEV₁ in the Control Group was 43.27% at baseline with a standard deviation of 17.44%. In the Intervention Group the mean %FEV₁ at baseline was 43.33% with a standard deviation of 21.15%. No significant difference in %FEV₁ was found at baseline between the Control and Intervention groups (p=0.98). These values were lower than expected since patients did not use the spirometer correctly. Oga et al (2002)⁸¹ reported a mean baseline %FEV₁ value of 71.60% with a standard deviation of 21.90%. This study was an investigation and comparison of the responsiveness of
health status scores in asthmatic patients during treatment using three different
disease-specific measures. The study included 170 patients with newly diagnosed
asthma over a six-month period. Patients underwent treatment with inhaled
corticosteroids in accordance with the guidelines. A health status evaluation using
three disease-specific measures, and pulmonary function tests were performed on the
initial visit, and at 3 months and 6 months. The effect-size and the standardised
response mean were used as responsiveness indexes. Again, the values obtained for
baseline %FEV₁ in the present study were much lower than those values obtained in
the study mentioned.

4.1.10 INHALER TECHNIQUE SCORES AT BASELINE

Inhaler technique was assessed using a seven point assessment scale previously
described in section 3.3 (page 51). The maximum that could be scored for inhaler
technique was seven. Patients would get one mark for each step if the action was
performed correctly.

The following graph indicates the baseline scores for inhaler technique in both the
Control and Intervention groups:

![Graph showing inhaler technique scores at baseline](image)

**Figure 4.18** Baseline inhaler technique scores in the Control
and Intervention groups (n=120)
The mean score for inhaler technique at baseline in the Control Group was 2.75 with a standard deviation of 1.23. In the Intervention Group the mean score for inhaler technique was 3.12 with a standard deviation of 1.26. No significant difference between the Control and Intervention groups for baseline inhaler technique was found (p=0.11).

Figure 4.19 illustrates the number of correct actions for each of the seven steps at baseline in the Control and Intervention groups:

![Figure 4.19 Baseline inhaler technique scores indicating correct actions in the Control and Intervention groups (n=120)](image)

Many patients did not exhale slowly before the onset of inhalation (step 2) and did not hold their breath for about ten seconds after inhalation (step 5). Almost all of the patients included in the study did not wait before the next inhalation (step 7) and used more than one puff per inhalation (step 6). This indicated that patients included in the present study did not use the inhaler correctly. Williams et al (1998)\(^5\) noted that self-management skills for example MDI ability, not just knowledge, are poorer among patients with limited reading skills as was the case in this study group. Only one patient managed to score a perfect 7 for baseline inhaler technique score. (See section 3.3 for each of the seven steps)

Berg et al (2004)\(^5\) noted that nearly 40% of all patients use their inhalers incorrectly. Even with a good technique, only about 10% of the medicine actually reaches the
lungs. When the inhaler is used incorrectly, an even smaller amount may reach the lungs. Patients included in this study were not using the metered dose inhaler correctly and it can therefore be assumed that the drug (especially the inhaled beclomethasone) did not produce the required effect since sub-therapeutic levels of the drug actually reached the lungs.

A spacer provides several advantages when recommended for use with a metered dose inhaler. These include less skill and coordination required for its use, an increase in the dose of medication reaching the lungs as well as a decrease in droplet size with the use of large volume spacers. For these reasons the use of a spacer in the form of a specially modified 500ml plastic bottle was recommended to 44 patients in the Intervention Group and the use of this spacer was explained. Due to the lack of portability of the customised spacer, it was only used together with the inhaled beclomethasone and not with the salbutamol inhaler. Patients therefore still required good inhaler technique to use the salbutamol inhaler. Upon returning for the follow-up interview, patients reported a positive attitude towards using these spacers.
Figure 4.20 summarises the asthma medication prescribed in the Control and Intervention groups pre-intervention:

![Graph showing medication regimens pre-intervention](image)

**Figure 4.20  Medication regimens pre-intervention (n=120)**

A total of 56 patients (96.33%) in the Control Group and 54 patients (90.00%) in the Intervention Group were prescribed theophylline tablets in the form of Sandoz-Theophylline® SR 250mg tablets.

Only 10 patients (16.67%) in the Control Group and 11 patients (18.33%) in the Intervention Group were using inhaled beclomethasone (Beclate® 50µg inhaler) and many of these patients indicated that they only used one puff (50µg) in the morning. This dosage is not sufficient since the recommended daily dose for intermittent asthma in adults is 200-500µg daily. A higher dose of inhaled corticosteroid MDI, for example beclomethasone 100µg instead of 50µg will also assist in drug delivery to the lungs since more of the active ingredient can be deposited in a single puff. This will then assist patients in the use of the MDI since fewer puffs will have to be used to deliver the required amount of active ingredient to the lungs. It should therefore be recommended that the Department of Health therefore look at including the Beclate® 100µg inhaler instead of the Beclate® 50µg on the EDL.
Chronic oral corticosteroid use can lead to a range of adverse effects which include osteoporosis, sodium and water retention, hyperglycaemia, hypokalemia, hypertension, skin striae, glaucoma and impaired wound healing. Systemic corticosteroids should therefore be reserved for cases where other therapies are unsuccessful, in severe asthma exacerbations or to prevent an impending episode of severe asthma. Chronic systemic corticosteroid use was therefore restricted as far as possible in patients in the Intervention Group. Prednisone use was stopped slowly by tapering the dose over a period of about 6 weeks. This was explained to the patients and instructions were written on the labels on how the prednisone dose should be tapered.

A total of 14 patients (23.33%) in the Control Group and 12 patients (20.00%) in the Intervention Group were also using chronic systemic corticosteroid therapy in the form of prednisone 5mg tablets. The prescribed dose was normally 15mg on Mondays, Wednesdays and Fridays. This was a matter of concern due to the severe side effects normally experienced with chronic systemic corticosteroid use. A few patients reported that they suffered heartburn and indigestion and this could be linked to the chronic corticosteroid use. Since hypertension was the most prevalent co-morbidity in the study population, chronic corticosteroid therapy is also questionable since corticosteroids should be used with caution in hypertensive patients due to the risk of sodium and water retention.

A total of 55 patients (91.67%) in the Control Group and 55 patients (91.67%) in the Intervention Group were prescribed inhaled salbutamol (Asthaven®). The normal dose of salbutamol is 100-200µg (1-2 puffs) three to four times daily. Some of the patients indicated that they would use more than two inhalers (300 doses per inhaler) per month. This equates to more than twenty puffs per day. This was an indication of poor management of their asthma and suggested a need for preventer drugs.

Recommended changes to medication regimens of patients in the Intervention Group were made in accordance with the National Guidelines for the Management of Asthma in Adults. Changes were only made after consultation with and acceptance by the clinic doctor. No problems were experienced in discussions with the doctor and he was very open to suggestions and enthusiastic to see the outcome of these changes.
Most of the interventions involved stopping sustained-release theophylline therapy or chronic prednisone use and starting inhaled corticosteroid therapy in the form of a beclomethasone 50µg inhaler. The recommended dose for most of the patients was 200µg twice daily. The patients in the Intervention Group were educated on the correct use of inhalers. The use of 500ml plastic bottles that were converted for use as a spacer was also suggested to all patients in the Intervention Group. They were educated on the correct use of these spacers and the importance of rinsing the mouth after use.

The most common asthma medication regimen was Sandoz-Theophylline® SR 250mg one tablet twice daily, together with Asthavent® (salbutamol) inhalation as required. According to the medication history in the patient files, the medication regimens of asthma patients were not often reviewed. According to the Essential Drugs List Guidelines, theophylline is not one of the recommended drugs in the treatment of asthma. On the other hand, the National Guidelines for the management of asthma in adults published by the Allergy Society of South Africa (ALLSA) recommends the use of theophylline only as an alternative to long-acting β2 agonists in the treatment of mild persistent asthma although long-acting β2 agonists are preferred to theophylline. The reason for using theophylline in the clinic instead of long-acting β2 agonists could be that the long-acting β2 agonists are not included on the EDL due to the high cost of the long-acting β2 agonists. For the treatment of moderate and severe persistent asthma, theophylline is indicated only as add-on therapy for patients on inhaled corticosteroid therapy and long-acting β2 agonists who fail to respond to treatment. The chronic use of theophylline in the management of asthma is therefore not the recommended treatment for the control of chronic asthma. Theophylline is not an ideal drug since it has a very narrow therapeutic range (5-15µg/ml) and close therapeutic drug monitoring is therefore needed since theophylline intoxication can result in cardiovascular problems such as tachycardia, hypotension and palpitations, as well as seizures and even death. Dose-dependent side effects that can be expected with theophylline use include nausea, gastrointestinal upset, headache, arrhythmias, tachycardia and convulsions. Drug interactions with theophylline are also very common since theophylline is extensively hepatically metabolised and theophylline plasma levels are influenced by any hepatic enzyme-inducing drugs such as carbamazepine, phenytoin, rifampicin and phenobarbitone or enzyme-inhibiting drugs.
such as allopurinol, calcium-channel blockers, isoniazid, and the macrolides. The use of chronic theophylline in the treatment of asthma in the Intervention Group was therefore minimised as far as possible.

A total of 49 prescribing recommendations were made in the Intervention Group. All prescribing recommendations were discussed with the clinic doctor prior to making any changes to the patient’s medication regimen. After the medication changes were accepted by the clinic doctor, a new prescription for the new medication regimen would be obtained for the patient from the doctor.

It was apparent during this study that many of the health care workers at the clinic did not know the importance of inhaled corticosteroids in the management of asthma. A method of encouraging health care professionals to adjust asthma medication where needed and to stress the importance of inhaled corticosteroid use, as well as the side-effects associated with chronic systemic corticosteroid use, should be developed as this will ultimately improve the management of asthmatic patients.

Not all patients accepted the prescribing recommendations made and some patients did not return for medication changes. If the clinic doctor was not on duty when prescribing recommendations were made, the patient had to return to the clinic for the prescription changes after prescribing recommendations had been discussed with the doctor and approval had been granted. This resulted in patients not returning for the follow-up visit for suggested medication changes even though these changes had been accepted by the doctor. Suggested changes could therefore not be implemented for these patients. This was only the case in two patients (3.00%) (n=60). Three patients (5.00%) (n=60) did not accept the suggested changes. All three patients did not want to stop theophylline treatment.
The following table provides an outline of the medication changes suggested in the Intervention Group:

### Table 4.2 Medication changes

<table>
<thead>
<tr>
<th>PATIENT NUMBER</th>
<th>MEDICATION PRIOR TO INTERVENTION</th>
<th>RECOMMENDATION</th>
<th>ACCEPTED?</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>- Theophylline SR 250mg bd&lt;br&gt; - Salbutamol inhaler prn</td>
<td>- Stop theophylline&lt;br&gt; - Start inhaled beclomethasone 200µg bd</td>
<td>Yes</td>
</tr>
<tr>
<td>2</td>
<td>- Theophylline SR 250mg bd&lt;br&gt; - Salbutamol inhaler prn&lt;br&gt; - Inhaled beclomethasone 200µg bd</td>
<td>None (patient’s asthma was controlled)</td>
<td>-</td>
</tr>
<tr>
<td>4</td>
<td>- Theophylline SR 250mg bd&lt;br&gt; - Salbutamol inhaler prn&lt;br&gt; - Inhaled beclomethasone 50µg bd&lt;br&gt; - Prednisone 15mg Mon, Wed, Fri</td>
<td>- Stop prednisone-taper&lt;br&gt; - Increase inhaled beclomethasone to 200µg bd</td>
<td>Yes</td>
</tr>
<tr>
<td>11</td>
<td>- Theophylline SR 250mg bd&lt;br&gt; - Salbutamol inhaler prn</td>
<td>Sent for TB test</td>
<td>Yes</td>
</tr>
<tr>
<td>12</td>
<td>- Theophylline SR 250mg bd&lt;br&gt; - Salbutamol inhaler prn</td>
<td>Sent for TB test</td>
<td>Yes</td>
</tr>
<tr>
<td>13</td>
<td>- Theophylline SR 250mg bd&lt;br&gt; - Salbutamol inhaler prn</td>
<td>- Stop theophylline&lt;br&gt; - Start inhaled beclomethasone 200µg bd</td>
<td>Yes</td>
</tr>
<tr>
<td>16</td>
<td>- Theophylline SR 250mg bd&lt;br&gt; - Salbutamol inhaler prn</td>
<td>- Stop theophylline&lt;br&gt; - Start inhaled beclomethasone 200µg bd</td>
<td>Yes</td>
</tr>
<tr>
<td>18</td>
<td>- Theophylline SR 250mg bd</td>
<td>Asthma medication stopped after consultation with doctor-dyspnoea caused by CCF, not asthma</td>
<td>Yes</td>
</tr>
<tr>
<td>19</td>
<td>- Theophylline SR 250mg bd&lt;br&gt; - Salbutamol inhaler prn</td>
<td>- Stop theophylline&lt;br&gt; - Start inhaled beclomethasone 200µg bd</td>
<td>No (patient did not accept)</td>
</tr>
<tr>
<td>Day</td>
<td>Medications</td>
<td>Instructions</td>
<td>Notes</td>
</tr>
<tr>
<td>-----</td>
<td>-------------</td>
<td>--------------</td>
<td>-------</td>
</tr>
</tbody>
</table>
| 20  | Salbutamol inhaler prn  
      | Start inhaled beclomethasone 100µg bd (intermittent asthma) | Yes |  
| 21  | Theophylline SR 250mg bd  
      | Salbutamol inhaler prn  
      | Inhaled beclomethasone 50µg bd (patient did not use this)  
      | Prednisone 10mg once weekly | Patient encouraged to use inhaled beclomethasone.  
      | Spacer recommended | - |
| 23  | Theophylline SR 250mg bd  
      | Salbutamol inhaler prn  
      | Inhaled beclomethasone 50µg bd  
      | Prednisone 10mg Mon, Wed, Fri | Stop prednisone-taper  
      | Increase inhaled beclomethasone to 200µg bd | Yes |
| 24  | Theophylline SR 250mg bd  
      | Salbutamol inhaler prn | Stop theophylline  
      | Start inhaled beclomethasone 200µg bd | Yes |
| 26  | Theophylline SR 250mg bd  
      | Salbutamol inhaler prn  
      | Prednisone 15mg Mon, Wed, Fri | Stop theophylline  
      | Stop prednisone-taper  
      | Start inhaled beclomethasone 200µg bd | Yes |
| 27  | Theophylline SR 250mg bd  
      | Salbutamol inhaler prn | Stop theophylline  
      | Start inhaled beclomethasone 200µg bd | Yes |
| 28  | Theophylline SR 250mg bd  
      | Salbutamol inhaler prn | Stop theophylline  
      | Start inhaled beclomethasone 200µg bd | Yes |
| 29  | Theophylline SR 250mg bd  
      | Salbutamol inhaler prn  
      | Prednisone 15mg Mon, Wed, Fri | Stop prednisone-taper  
      | Start inhaled beclomethasone 200µg bd | Yes  
      | (Patient did not return for medication changes) |
| 32  | Theophylline SR 250mg bd  
      | Salbutamol inhaler prn | None (Patient was on TB treatment) | - |
| 33  | Theophylline SR 250mg bd  
      | Salbutamol inhaler prn | Stop theophylline  
<pre><code>  | Start inhaled beclomethasone 200µg bd | Yes |
</code></pre>
<table>
<thead>
<tr>
<th>Day</th>
<th>Medication Changes</th>
</tr>
</thead>
</table>
| 35  | o Salbutamol inhaler prn  
   o Prednisone 15mg Mon,Wed,Fri  
   o Start inhaled beclomethasone 200µg bd  
   o Atenolol also stopped | Yes |
| 37  | o Theophylline SR 250mg bd  
   o Salbutamol inhaler prn  
   o Prednisone 15mg Mon,Wed,Fri  
   o Stop theophylline  
   o Stop prednisone-taper  
   o Start inhaled beclomethasone 200µg bd | Yes |
| 38  | o Theophylline SR 250mg bd  
   o Salbutamol inhaler prn  
   o Inhaled beclomethasone 50µg bd  
   o Increase inhaled beclomethasone 200µg bd | Yes |
| 39  | o Theophylline SR 250mg bd  
   o Salbutamol inhaler prn  
   o Prednisone 15mg Mon,Wed,Fri  
   o Stop prednisone-taper  
   o Start inhaled beclomethasone 200µg bd | Yes |
| 42  | o Theophylline SR 250mg bd  
   o Salbutamol inhaler prn  
   o Stop theophylline  
   o Start inhaled beclomethasone 200µg bd | Yes  
(Patient did not return for medication changes) |
| 45  | o Salbutamol inhaler prn  
   o Inhaled beclomethasone 50µg bd  
   o Increase inhaled beclomethasone 200µg bd | Yes |
| 46  | o Theophylline SR 250mg bd  
   o Salbutamol inhaler prn  
   o Stop theophylline  
   o Start inhaled beclomethasone 200µg bd | Yes |
| 53  | o Theophylline SR 250mg bd  
   o Salbutamol inhaler prn  
   o Stop theophylline  
   o Start inhaled beclomethasone 200µg bd | Yes |
| 56  | o Theophylline SR 250mg bd  
   o Salbutamol inhaler prn  
   o Prednisone 15mg Mon,Wed,Fri  
   o Stop prednisone-taper  
   o Start inhaled beclomethasone 200µg bd  
   o Patient did not accept | No |
| 59  | o Theophylline SR 250mg bd  
   o Salbutamol inhaler prn  
   o Stop theophylline  
   o Start inhaled beclomethasone 200µg bd | Yes |
<table>
<thead>
<tr>
<th>No</th>
<th>Theophylline SR 250mg bd</th>
<th>Salbutamol inhaler prn</th>
<th>Start inhaled beclomethasone 200µg bd</th>
<th>Prednisone 15mg Mon,Wed,Fri</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>60</td>
<td>o</td>
<td>o</td>
<td>o</td>
<td></td>
<td></td>
</tr>
<tr>
<td>63</td>
<td>None (has not attended clinic in a long time)</td>
<td>o</td>
<td>o</td>
<td></td>
<td></td>
</tr>
<tr>
<td>68</td>
<td>o</td>
<td>o</td>
<td>o</td>
<td></td>
<td></td>
</tr>
<tr>
<td>70</td>
<td>o</td>
<td>o</td>
<td>None ( after consultation with doctor: emphysema rather than asthma)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>71</td>
<td>o</td>
<td>o</td>
<td>None (patient’s asthma was controlled)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>75</td>
<td>o</td>
<td>o</td>
<td>o</td>
<td></td>
<td></td>
</tr>
<tr>
<td>77</td>
<td>o</td>
<td>o</td>
<td>o</td>
<td></td>
<td></td>
</tr>
<tr>
<td>81</td>
<td>None</td>
<td></td>
<td>Patient sent to doctor for consultation and prescription (unknown outcome)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>83</td>
<td>o</td>
<td>o</td>
<td>o</td>
<td></td>
<td></td>
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<td>85</td>
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<td>o</td>
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<td></td>
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<td>86</td>
<td>o</td>
<td>o</td>
<td>o</td>
<td></td>
<td></td>
</tr>
<tr>
<td>88</td>
<td>o</td>
<td>o</td>
<td>o</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>Theophylline SR 250mg bd</td>
<td>Salbutamol inhaler prn</td>
<td>Inhaled beclomethasone 50µg bd</td>
<td>Prednisone 15mg Mon,Wed,Fri</td>
<td>Status</td>
</tr>
<tr>
<td>-----</td>
<td>--------------------------</td>
<td>------------------------</td>
<td>-------------------------------</td>
<td>-----------------------------</td>
<td>--------</td>
</tr>
<tr>
<td>89</td>
<td>Stop theophylline</td>
<td>Start inhaled</td>
<td>beclomethasone 200µg bd</td>
<td>None</td>
<td>Yes</td>
</tr>
<tr>
<td>97</td>
<td>Stop theophylline</td>
<td>Start inhaled</td>
<td>beclomethasone 200µg bd</td>
<td>None</td>
<td>Yes</td>
</tr>
<tr>
<td>98</td>
<td>None (patient’s asthma was controlled)</td>
<td></td>
<td></td>
<td>None</td>
<td>-</td>
</tr>
<tr>
<td>100</td>
<td>None (patient had lung damage due to PTB according to Xrays)</td>
<td></td>
<td></td>
<td>None</td>
<td>-</td>
</tr>
<tr>
<td>101</td>
<td>Stop theophylline</td>
<td>Start inhaled</td>
<td>beclomethasone 200µg bd</td>
<td>None</td>
<td>Yes</td>
</tr>
<tr>
<td>102</td>
<td>Stop theophylline</td>
<td>Start inhaled</td>
<td>beclomethasone 200µg bd</td>
<td>None</td>
<td>Yes</td>
</tr>
<tr>
<td>103</td>
<td>Stop theophylline</td>
<td>Start inhaled</td>
<td>beclomethasone 200µg bd</td>
<td>None</td>
<td>Yes</td>
</tr>
<tr>
<td>104</td>
<td>Stop theophylline</td>
<td>Start inhaled</td>
<td>beclomethasone 200µg bd</td>
<td>None</td>
<td>Yes</td>
</tr>
<tr>
<td>105</td>
<td>Stop theophylline</td>
<td>Start inhaled</td>
<td>beclomethasone 200µg bd</td>
<td>None</td>
<td>Yes</td>
</tr>
<tr>
<td>107</td>
<td>Stop theophylline</td>
<td>Increase inhaled</td>
<td>beclomethasone to 200µg bd</td>
<td>None</td>
<td>Yes</td>
</tr>
<tr>
<td>108</td>
<td>Start inhaled</td>
<td>beclomethasone 200µg bd</td>
<td></td>
<td>None</td>
<td>Yes</td>
</tr>
<tr>
<td>109</td>
<td>Stop theophylline</td>
<td>Start inhaled</td>
<td>beclomethasone 200µg bd</td>
<td>None</td>
<td>Yes</td>
</tr>
<tr>
<td></td>
<td>Medication Changes</td>
<td>None</td>
<td>Action Taken</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>-------------------</td>
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<td>--------------</td>
<td>-----</td>
<td></td>
</tr>
<tr>
<td>110</td>
<td>Theophylline SR 250mg bd</td>
<td>None (patient’s asthma was controlled)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Salbutamol inhaler prn</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Inhaled beclomethasone 50µg bd</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>113</td>
<td>Theophylline SR 250mg bd</td>
<td>None (doctor suspected that the patient had COPD, not asthma)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Salbutamol inhaler prn</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>115</td>
<td>Theophylline SR 250mg bd</td>
<td>None (doctor suspected that the patient had COPD, not asthma)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Salbutamol inhaler prn</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>116</td>
<td>Theophylline SR 250mg bd</td>
<td>Stop theophylline, Stop prednisone-taper</td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Salbutamol inhaler prn</td>
<td>Stop theophylline</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Inhaled beclomethasone 50µg bd</td>
<td>Stop prednisone-taper</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Prednisone 15mg Mon,Wed,Fri</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>117</td>
<td>Theophylline SR 250mg bd</td>
<td>Start inhaled beclomethasone 200µg bd</td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>118</td>
<td>Theophylline SR 250mg bd</td>
<td>Stop theophylline</td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Salbutamol inhaler prn</td>
<td>Start inhaled beclomethasone 200µg bd</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>120</td>
<td>Theophylline SR 250mg bd</td>
<td>Stop theophylline</td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Salbutamol inhaler prn</td>
<td>Stop theophylline</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Figure 4.21 depicts the number of patients who returned and accepted the medication changes as well as the number of prescribing changes accepted by the clinic doctor:

![Figure 4.21 Recorded medication changes (n=60)](image-url)
The interventions and cases were much more complicated than what is indicated in Table 4.2. The following two case studies illustrate two typical interventions:

Patient A, a 43 year old female with mild asthma, was using theophylline SR tablets 250mg twice daily and inhaled salbutamol as needed. She was randomly allocated to the Intervention Group and her medication was changed after consultation with the doctor to inhaled beclomethasone 200 µg twice daily with the use of a spacer in the form of a converted 500ml plastic bottle and inhaled salbutamol as needed. The theophylline SR use was stopped. Her %PEFR increased from 8.00% at the first appointment to 17.00% at the second visit. Both the %FEV and %FEV₁ values increased from 15.00% to 33.00%. The values for her %PEFR, %FVC and %FEV₁ were low since the patient could not close her mouth properly due to a case of Bell’s Palsy two years ago and the spirometer could therefore not take accurate readings. Her total score for the KASE-AQ increased from 151 at baseline to 156 post-intervention, although her average QOL measured by the AQLQ dropped from 3.5 at baseline to 3.16 post-intervention.

Patient B, female 45 years old with moderate persistent asthma was using theophylline SR 250 mg twice daily and inhaled salbutamol as needed. She was also randomly allocated to the Intervention Group and her medication regimen changed to the following after consultation with the doctor: inhaled beclomethasone 200 µg twice daily and inhaled salbutamol as needed. She was also given a 500ml plastic bottle spacer and educated on the correct use of inhalers and spacers. Her %PEFR decreased from 44.00% to 35.00% while her %FVC decreased from 73.00% to 23.00% and her %FEV₁ from 78.00% to 37.00%. The reason for this decrease in spirometric values could be that she had an upper respiratory tract infection at the time when the second interview was conducted. Her average QOL score increased from 1.59 at baseline to 2.44 post-intervention and her total KASE-AQ score increased from 140 to 179.
4.3 POST-INTERVENTION ASSESSMENT

The post-intervention assessment was conducted three months after the initial interviews.

The following table summarises the post-intervention data of the participants in the study:

Table 4.3 Post-intervention scores of participants included in the study

<table>
<thead>
<tr>
<th></th>
<th>Control Group (n=42)</th>
<th>Intervention Group (n=40)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>QOL questionnaire (SD):</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Activity limitation (best=7)</td>
<td>2.33 (0.65)</td>
<td>2.69 (0.87)</td>
</tr>
<tr>
<td>Symptoms (best=7)</td>
<td>2.55 (0.91)</td>
<td>2.76 (1.17)</td>
</tr>
<tr>
<td>Emotional function (best=7)</td>
<td>2.03 (0.95)</td>
<td>2.26 (1.32)</td>
</tr>
<tr>
<td>Environmental stimuli (best=7)</td>
<td>1.29 (0.67)</td>
<td>1.49 (0.79)</td>
</tr>
<tr>
<td>Total (max=224)</td>
<td>71.57 (20.76)</td>
<td>79.97 (29.21)</td>
</tr>
<tr>
<td>Average (max=7)</td>
<td>2.24 (0.65)</td>
<td>2.50 (0.91)</td>
</tr>
<tr>
<td><strong>KASE-AQ (SD):</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knowledge (max=20)</td>
<td>10.64 (2.88)</td>
<td>12.03 (3.03)</td>
</tr>
<tr>
<td>Attitude (max=100)</td>
<td>79.38 (6.72)</td>
<td>79.03 (7.65)</td>
</tr>
<tr>
<td>Self-efficacy (max=100)</td>
<td>70.38 (8.53)</td>
<td>71.83 (8.71)</td>
</tr>
<tr>
<td>Total (max=220)</td>
<td>160.40 (12.76)</td>
<td>162.88 (15.26)</td>
</tr>
<tr>
<td>%PEFR mean (SD)</td>
<td>28.57 (14.49)</td>
<td>29.75 (15.01)</td>
</tr>
<tr>
<td>%FVC mean (SD)</td>
<td>42.57 (16.78)</td>
<td>48.35 (19.38)</td>
</tr>
<tr>
<td>%FEV1 mean (SD)</td>
<td>43.12 (17.35)</td>
<td>47.73 (20.12)</td>
</tr>
<tr>
<td>Inhaler technique (max=7)</td>
<td>3.02 (1.12)</td>
<td>4.45 (1.24)</td>
</tr>
</tbody>
</table>

As can be seen from the above table, patients in the Intervention Group scored higher in all categories of both the QOL and KASE-AQ questionnaire compared with the Control Group, except for the attitude subscale section of the KASE-AQ. The scores for most of the variables in the Control Group decreased from baseline to three months. However, the scores for the attitude subscale of KASE-AQ, %PEFR and inhaler technique improved from baseline to three months in the Control Group. The reason for the improvement in attitude could be that patients’ attitudes towards their disease improved because they were spending time discussing their disease, symptoms and medication.
4.3.1 OUTLIERS

The data were analysed for outliers and the following graph indicates the one outlier that was identified:

![Graph showing total QOL vs pre-intervention score with an outlier at 84 (average=2.63) and 196 (average=6.13) post-intervention.]

Figure 4.22 Outlier for post-intervention scores

From the above graph it can be seen that there was one outlier with the post-intervention scores (see I at top of graph). This particular patient went from a total score of 84 (average=2.63) for the QOL questionnaire to a total score of 196 (average=6.13) post-intervention. She also scored 141 for the KASE-AQ pre-intervention and 179 post-intervention. Both her %FVC and %FEV₁ increased significantly, although her %PEFR decreased. During the statistical analysis of the results this outlier was identified and after revisiting the data to ensure validity, it was included in the statistical analysis. This particular patient was very positive about the improvement she experienced with regards to her asthma management post-intervention.
4.3.2 REASONS FOR NOT USING PAIRED T-TEST RESULTS

Paired t-test analyses of the baseline and post-intervention scores for both the Control and Intervention groups were conducted. The following table outlines the results of the paired t-tests:

Table 4.4 Paired t-test results

<table>
<thead>
<tr>
<th>Variable</th>
<th>CONTROL GROUP</th>
<th>INTERVENTION GROUP</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>t-value</td>
<td>p-value</td>
</tr>
<tr>
<td></td>
<td>Mean of</td>
<td></td>
</tr>
<tr>
<td></td>
<td>differences</td>
<td></td>
</tr>
<tr>
<td>Total QOL</td>
<td>1.485</td>
<td>0.1451</td>
</tr>
<tr>
<td>Activity limitation</td>
<td>2.950</td>
<td>0.005228*</td>
</tr>
<tr>
<td>Symptoms</td>
<td>-0.5057</td>
<td>0.6158</td>
</tr>
<tr>
<td>Emotional function</td>
<td>0.7807</td>
<td>0.4395</td>
</tr>
<tr>
<td>Environmental stimuli</td>
<td>2.715</td>
<td>0.009664*</td>
</tr>
<tr>
<td>KASE-AQ Total</td>
<td>0.8992</td>
<td>0.3738</td>
</tr>
<tr>
<td>Knowledge</td>
<td>2.115</td>
<td>0.04052*</td>
</tr>
<tr>
<td>Attitude</td>
<td>-0.8047</td>
<td>0.4256</td>
</tr>
<tr>
<td>Self-efficacy</td>
<td>1.281</td>
<td>0.2073</td>
</tr>
<tr>
<td>%PEFR</td>
<td>0.9271</td>
<td>0.3593</td>
</tr>
<tr>
<td>%FVC</td>
<td>3.189</td>
<td>0.002732*</td>
</tr>
<tr>
<td>%FEV₁</td>
<td>1.718</td>
<td>0.09333</td>
</tr>
<tr>
<td>Inhaler technique</td>
<td>-1.776</td>
<td>0.08317</td>
</tr>
</tbody>
</table>

* Indicates statistically significant values (p<0.05)

The problem was that for many of the variables, the Control and Intervention groups were not evenly matched with regards to their baseline values, and this could have created a confounding effect.
Table 4.5 outlines an example of a paired t-test for the attitude subscale section of the KASE-AQ:

**Table 4.5  Example of a paired t-test for attitude scores**

<table>
<thead>
<tr>
<th>Paired t-test</th>
<th>INTERVENTION GROUP</th>
<th>CONTROL GROUP</th>
</tr>
</thead>
<tbody>
<tr>
<td>t = -4.054, df = 39, p-value = 0.0002328</td>
<td>t = -0.8047, df = 41, p-value = 0.4256</td>
<td></td>
</tr>
<tr>
<td>mean of the differences 5.575</td>
<td>mean of the differences 1.143</td>
<td></td>
</tr>
</tbody>
</table>

From the above it can be seen that the paired t-test shows a statistically significant difference for the Intervention Group (p=0.0002328) with an average increase in the attitude score of 5.575, while there is an insignificant result for the Control Group (p=0.4256) with an average increase in attitude score of 1.143.

The following graph illustrates the change in attitude score for the Intervention Group:

**Figure 4.23  Change in attitude score for the Intervention Group**
It can be seen from the above graph that for the Intervention Group there appears to be a negative relationship between the change in attitude score and the pre-intervention attitude score. It was observed that patients who initially had a lower pre-intervention attitude score improved more than, for example patients with a 65 pre-intervention score, who tended to score about 10 more points post-intervention (from 65 to 75). Patients who initially had a high pre-intervention score on the other hand, did not improve as much. For example, on average, a patient with an 80 pre-intervention score did not score any higher on their post-intervention score. This negative relationship also holds for the Control Group, the graph for which is plotted below.

![Change in Attitude Score Control Group](figure424.png)

**Figure 4.24  Change in attitude score for the Control Group**

Once again the marked negative relationship can be seen, where people with low pre-intervention attitude scores, tended to show a far greater improvement in attitude score than those who had started with high pre-intervention scores. This is illustrated by the regression line which is plotted in both graphs.
If the Control Group is compared with the Intervention Group, it was observed that the Control Group had many more patients who started off with high pre-intervention scores (>85), whereas there were no patients who started off with a high (>85) pre-intervention score in the Intervention Group.

To account for the baseline differences and this negative relationship, regression modelling in the form of ANCOVA was used to analyse differences between pre- and post-intervention scores. ANCOVA takes into account a subject’s pre-intervention score in the analysis and can compensate for baseline differences in the two groups.

This explains why the paired t-test showed an improvement in the Intervention Group and no improvement in the Control Group, even though both groups showed no difference when analysed by ANCOVA. There were more patients who started off from lower baseline values in the Intervention Group and, purely due to the negative relationship that existed between pre-intervention and post-intervention scores. This meant that their improvement was apparently greater than those in the Control Group who started off with higher baseline values.

Most of the subscales had similar graphs, where there tended to be a lot more patients in the Control Group with higher baseline values compared with the Intervention Group. This meant that the paired t-tests were often seemingly significant. However due to the fact that the Intervention Group initially had lower baseline values, they thus improved more than their Control counterparts who started off with higher scores.
4.3.4 CHANGES IN OUTCOME MEASURES AT THREE MONTHS AFTER RANDOMISATION

The following table outlines the changes in outcome measures at three months after randomisation:

Table 4.6 Changes in outcome measures at three months after randomisation

<table>
<thead>
<tr>
<th>Point increase</th>
<th>P value for group difference (ANCOVA)</th>
<th>Adjusted means</th>
<th>Cohen’s $d$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Intervention</td>
<td>Control</td>
<td></td>
</tr>
<tr>
<td>Total-QOL</td>
<td>12.067</td>
<td>0.02332*</td>
<td>81.85</td>
</tr>
<tr>
<td>Average-QOL</td>
<td>0.376</td>
<td>0.02369*</td>
<td>2.558</td>
</tr>
<tr>
<td>Activity limitations</td>
<td>0.427</td>
<td>0.01049*</td>
<td>2.726</td>
</tr>
<tr>
<td>Symptoms</td>
<td>-</td>
<td>0.10750</td>
<td>-</td>
</tr>
<tr>
<td>Emotional function</td>
<td>-</td>
<td>0.09864</td>
<td>-</td>
</tr>
<tr>
<td>Environmental</td>
<td>-</td>
<td>0.1081</td>
<td>-</td>
</tr>
<tr>
<td>Total-KASE-AQ</td>
<td>-</td>
<td>0.0607</td>
<td>-</td>
</tr>
<tr>
<td>Knowledge†</td>
<td>1.38</td>
<td>0.037*</td>
<td>-</td>
</tr>
<tr>
<td>Attitude†</td>
<td>-</td>
<td>0.82</td>
<td>-</td>
</tr>
<tr>
<td>Self-efficacy</td>
<td>-</td>
<td>0.0716</td>
<td>-</td>
</tr>
<tr>
<td>%PEFR</td>
<td>-</td>
<td>0.28574</td>
<td>-</td>
</tr>
<tr>
<td>%FVC</td>
<td>5.4691</td>
<td>0.022*</td>
<td>48.18</td>
</tr>
<tr>
<td>%FEV₁</td>
<td>4.7447</td>
<td>0.043*</td>
<td>47.80</td>
</tr>
<tr>
<td>Inhaler technique</td>
<td>1.2537</td>
<td>7.3x10⁻⁵*</td>
<td>4.362</td>
</tr>
</tbody>
</table>

* Indicates the statistically significant results with a p-value<0.05
† The baseline values for these two variables had no significant effect on the post-intervention values; ANOVA was therefore used instead of ANCOVA
4.3.4  QUALITY OF LIFE QUESTIONNAIRE SCORES

4.3.4.1  Total QOL scores

The following graph outlines the baseline and post-intervention mean scores for total QOL in the Control and Intervention groups:

![Graph showing baseline and post-intervention mean scores for total QOL](image)

**Figure 4.25  Baseline and post-intervention means for total QOL score**

* Indicates a statistically significant improvement (p<0.05)

The mean total QOL score increased from 69.80 (maximum=224) with a standard deviation of 22.67 at baseline, to 79.97 with a standard deviation of 29.21 post-intervention in the Intervention Group. In the Control Group the mean total QOL score dropped from 79.12 with a standard deviation of 20.94 at baseline, to 71.57 with a standard deviation of 20.76 post-intervention. The adjusted mean increase was 12.067. An analysis of covariance (ANCOVA) produced a p-value of 0.02332 which indicates a significant increase in total QOL in the Intervention Group compared with the Control Group. Cohen’s $d$ was calculated as 0.4782 which is indicative of a small effect.
4.3.4.3 Average QOL scores

The following graph outlines the baseline and post-intervention mean scores for average QOL in the Control and Intervention groups:

![Graph showing baseline and post-intervention mean scores for average QOL in Control and Intervention groups.]

**Figure 4.26 Baseline and post-intervention means for average QOL score**

* Indicates a statistically significant improvement (p<0.05)

An increase in average QOL score from 2.18 of a maximum possible score of 7 with a standard deviation of 0.71 to 2.50 with a standard deviation of 0.91 in the Intervention Group was observed. The Control Group showed a decrease in average QOL score from 2.47 with a standard deviation of 0.65 at baseline, to 2.24 with a standard deviation of 0.65 post-intervention. A p-value of 0.02369 was calculated using ANCOVA and this indicated a statistically significant impact on average QOL since the p-value was less than 0.05. Patients in the Intervention Group therefore experienced a significant increase in average quality of life due to the intervention. Cohen’s $d$ was calculated as 0.4768. The adjusted mean increase was 0.376.

According to Juniper et al (1999)** a change in score of 0.5 on the seven point scale is considered clinically meaningful and is regarded as the minimal important difference (MID) (refer section 2.3.2). The mean average QOL score of patients in the Intervention Group only increased by 0.32 and this can therefore not be regarded as clinically meaningful according to the Juniper MID even though the p-value was less than 0.05.
Juniper *et al* (2005)\(^7\) recorded a post-intervention average QOL score of 5.53, which indicated an increase in average QOL of 0.58 from the baseline mean average QOL score of 4.95. The study by Kheir *et al* (2001)\(^9\) showed a 0.3 average point increase for average QOL in patients in their intervention group with a post-intervention mean average QOL score of 5.1 (SD= 1.2). Patients in the Intervention Group in the present study therefore showed a similar increase in average QOL compared with the study by Kheir *et al* even though the increase in mean average quality of life was not clinically meaningful.

### 4.3.4.3 Activity limitations scores

The following graph outlines the baseline and post-intervention mean scores for activity limitation in the Control and Intervention groups:

![Graph showing activity limitations scores](image.png)

**Figure 4.27** Baseline and post-intervention means for activity limitation score

* Indicates a statistically significant improvement (p<0.05)

The mean activity limitation score in the Intervention Group increased from 2.54 (maximum possible score of 7) with a standard deviation of 0.63 at baseline, to 2.69 with a standard deviation of 0.87 post-intervention. The Control Group showed a decrease in mean activity limitation from 2.7 with a standard deviation of 0.69 at baseline to 2.33 with a standard deviation of 0.65 post-intervention. ANCOVA produced a p-value of 0.01049 which indicated a statistically significant increase in mean activity limitation score in the Intervention Group with an adjusted mean increase of 0.427. This would suggest that the intervention therefore had a positive
impact on the quality of life regarding activity limitation of patients. Statistically, ANCOVA showed a significant impact on QOL, however the intervention only showed a 0.15 increase in average activity limitation score in the Intervention Group and this was less than the MID of 0.5 determined by Juniper et al.\textsuperscript{42} The intervention therefore showed a statistically significant change but not necessarily a clinically meaningful increase. Cohen’s $d$ was calculated as 0.5594 which indicated a moderate effect.

Juniper et al (2005)\textsuperscript{77} recorded a mean post-intervention activity limitation score of 5.61 which was a 0.52 increase from the baseline score of 5.09. The study by Kheir et al (2001)\textsuperscript{9} recorded a mean increase in activity limitation score in the Intervention Group of 0.3. The improvement observed in mean activity limitation score for patients included in the Intervention Group for the present study was slightly less and was recorded as 0.15. Patients in the Control Group showed a mean 0.34 decrease in activity limitation score. This meant that the intervention improved patients’ quality of life with regards to activity limitation due to asthma.

4.3.4.4 Symptoms scores

The baseline and post-intervention mean scores for symptoms are illustrated below:

![Figure 4.28 Baseline and post-intervention means for symptoms score](image)

In the Intervention Group the mean symptoms score increased from 2.20 (maximum possible score of 7) with a standard deviation of 0.95 at baseline, to a post-
intervention score of 2.76 with a standard deviation of 1.17. The Control Group showed a slight decrease in mean symptoms score from 2.61 with a standard deviation of 0.87 at baseline, to 2.55 with a standard deviation of 0.91 post-intervention. A p-value of 0.10750 was calculated using ANCOVA indicating that the increase was not statistically significant.

Juniper et al (2005) showed an increase in mean symptoms score of 0.65. An increase in mean symptoms score of 0.4 in the Intervention Group was recorded in 2001 by Kheir et al (2001). The patients in the present study therefore showed a slightly larger increase in mean symptoms score compared with the patients included in the Kheir et al study, since a mean change of 0.56 was observed in the Intervention Group. This would indicate a clinically meaningful increase in QOL (since MID=0.5) even though the increase was not statistically significant. The Control Group had a slight decrease in mean symptoms score of 0.06.

4.3.4.7 Emotional function scores

The following graph illustrates the baseline and post-intervention mean scores for emotional function in the Control and Intervention groups:

![Figure 4.29 Baseline and post-intervention means for emotional function score](image)

The mean emotional function score in the Intervention Group increased from 1.87 (maximum possible score of 7) with a standard deviation of 0.85 at baseline, to a post-intervention score of 2.26 with a standard deviation 1.32. In the Control Group the
mean emotional function score dropped from a baseline score of 2.28 with a standard deviation of 1.05, to a post-intervention score of 2.03 with a standard deviation of 0.95. Using ANCOVA a p-value of 0.09864 was calculated. Since the p-value was greater than 0.05, this increase in emotional function score was not statistically significant and the intervention therefore did not have a statistically significant impact on patient quality of life regarding emotional function. The change in emotional function score of 0.39 in the Intervention Group was also less than the MID of 0.5 and can therefore not be regarded as being clinically meaningful.

The study by Juniper et al (2005)\textsuperscript{77} showed a 0.57 increase in mean emotional function score in the Intervention Group. This increase is greater than the increase in mean emotional function score recorded in the present study. The study by Kheir et al (2001)\textsuperscript{9} showed a mean increase in emotional function score of 0.4 in the Intervention Group. This is very close to the mean increase in emotional function score of 0.39 observed in the Intervention Group for the present study. The Control Group showed a decrease in mean emotional function of 0.25.

\subsection{4.3.4.8 Environmental stimuli scores}

Figure 4.30 illustrates the baseline and post-intervention mean scores for environmental stimuli in the Control and Intervention groups:

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{environmental_stimuli_scores.png}
\caption{Baseline and post-intervention means for environmental stimuli score}
\end{figure}
A decrease in environmental stimuli score from a preintervention score of 1.52 (maximum score of 7) with a standard deviation of 0.70 at baseline, to a post-intervention score of 1.49 with a standard deviation of 0.79 was observed in the Intervention Group. The Control Group also showed a decrease in environmental stimuli score from 1.65 (preintervention) with a standard deviation of 0.71 at baseline, to 1.29 with a standard deviation of 0.67 post-intervention. Using ANCOVA, a p-value of 0.1081 was calculated, which showed that the intervention did not have a statistically significant effect on quality of life regarding environmental stimuli.

The slight decrease of 0.03 in mean environmental stimuli score does not coincide with the 0.2 point increase in mean environmental stimuli score observed in the study by Kheir et al (2001) or the 0.54 increase in the Juniper et al (2005) study. Possible reasons for this could be the time of year that the present study was conducted (March to August). Since cooler weather exacerbates asthma and the follow-up interviews were conducted in winter, this could explain why patients reported a negative effect on quality of life due to environmental stimuli. In these colder months the incidence of respiratory tract infections also increases which can also lead to asthma exacerbations.
4.3.5 KNOWLEDGE, ATTITUDE AND SELF-EFFICACY SCORES

4.4.5.1 Total KASE-AQ scores

The graph below illustrates the baseline and post-intervention mean scores for total KASE-AQ in the Control and Intervention groups:

![Graph showing baseline and post-intervention mean scores for total KASE-AQ](image)

**Figure 4.31 Baseline and post-intervention means for total KASE-AQ score**

The mean total KASE-AQ score increased from 152.32 of a maximum score of 220 with a standard deviation of 10.03 at baseline in the Intervention Group, to a post-intervention score of 162.88 with a standard deviation of 15.26. The Control Group showed a decrease in mean total KASE-AQ from 161.33 with a standard deviation of 16.92 at baseline, to a score of 160.40 with a standard deviation of 12.76 post-intervention. A p-value of 0.0607 was calculated using ANCOVA and this indicated that the intervention did not have a statistically significant impact on total KASE-AQ since the p-value was greater than 0.05.
4.4.5.2 Knowledge subscale of KASE-AQ scores

Illustrated below are the baseline and post-intervention mean scores for knowledge:

![Knowledge score graph](image)

**Figure 4.32 Baseline and post-intervention means for knowledge score**

* Indicates a statistically significant improvement (p<0.05)

The mean knowledge in the Intervention Group increased from a score of 10.78 of a maximum score of 20 with a standard deviation of 2.92 at baseline, to a post-intervention score of 12.03 with a standard deviation of 3.03. In the Control Group, the baseline score of 12.32 with a standard deviation of 4.23 decreased to a score of 10.64 with a standard deviation of 2.88 post-intervention. The adjusted mean increase was 1.38.

Since the baseline values for these two variables had no significant effect on the post-intervention values, analysis of variance (ANOVA) was used instead of ANCOVA in analysing the data. A p-value of 0.037 was calculated which indicated a statistically significant increase since the p-value was less than 0.05. The intervention therefore had a significant impact on knowledge in the Intervention Group with an average increase in knowledge score of 1.25. Cohen’s $d$ was calculated as 0.4677 which indicated a small effect.

The study by Put *et al* (2002) showed an increase in mean knowledge score from 11.8 with a standard deviation of 2.6 at baseline to a post-intervention mean of 16.4 with a standard deviation of 0.5. The patients included in the present study therefore showed a lower mean score for knowledge both at baseline and post-intervention and...
also showed a smaller increase in mean knowledge score compared with the patients included in the Put et al study. A possible reason for this could be the low level of literacy of the patients at the clinic. Patients had particular difficulty in naming the different components of the respiratory system. Williams et al (1998) found that inadequate literacy was strongly correlated with poorer knowledge of asthma and improper MDI use in a group of 483 patients with asthma, as was the case in this study.

A study of this nature therefore can have a positive impact on patient knowledge regarding asthma and ultimately improve the control of the patient’s condition since numerous studies demonstrated a marked improvement in asthma management and control with patient education. Education should be specifically aimed at patients with a low level of literacy and visual aids such as pictures should be used where possible. Future studies can possibly look at the impact of having monthly asthma group meetings at the clinic, aimed at asthma education.

4.4.5.3 Attitude subscale of KASE-AQ scores

The following graph illustrates baseline and post-intervention mean scores for attitude in the Control and Intervention groups:

![Graph showing baseline and post-intervention means for attitude score](image)

**Figure 4.33 Baseline and post-intervention means for attitude score**

In the Intervention Group, the mean attitude score increased from 73.98 (maximum possible score of 100) with a standard deviation of 5.56 at baseline, to 79.03 with a
standard deviation of 7.65 post-intervention. The Control Group also showed an increase in attitude score from 77.82 with a standard deviation of 8.64 at baseline, to a post-intervention score of 79.38 with a standard deviation of 6.72. Since the baseline values for these two variables had no significant effect on the post-intervention values, ANOVA was used again instead of ANCOVA in analysing the data. A p-value of 0.82 was calculated. This indicated that the increase in score was not statistically significant since the p-value was greater than 0.05.

An increase in mean attitude score from a score of 77.0 with a standard deviation of 4.0 at baseline to a post-intervention score of 88.0 with a standard deviation of 6.0 was documented by Put et al (2002). Again, patients in the present study scored lower for mean attitude both at baseline and post-intervention and the increase in mean attitude score was also smaller compared with the patients included in the study by Put et al. The patients included in the present study therefore did show an improvement in attitude regarding their disease even though this increase in attitude score was not statistically significant.

4.4.5.4 Self-efficacy subscale of KASE-AQ scores

Illustrated below are the baseline and post-intervention mean scores for self-efficacy:

![Graph showing baseline and post-intervention mean scores for self-efficacy]

The Intervention Group showed an increase in mean self-efficacy score from 67.55 (maximum possible score of 100) with a standard deviation of 6.51 at baseline, to a
post-intervention score of 71.83 with a standard deviation of 8.71. In the Control Group there was a decrease in mean self-efficacy score from 71.20 with a standard deviation of 9.13 at baseline, to a score of 70.38 with a standard deviation of 8.53 post-intervention. Using ANCOVA a p-value of 0.0716 was calculated. Since the p-value was greater than 0.05, this increase in self-efficacy in the Intervention Group was not statistically significant.

The study by Put et al (2002) documented a baseline mean self-efficacy score of 71.0 with a standard deviation of 8.0 and a post-intervention mean self-efficacy score of 85.0 with a standard deviation of 10.0. Patients in the present study therefore scored lower at baseline and post-intervention for mean self-efficacy score and the increase in mean self-efficacy score was not as noticeable as for the Put et al study. Patients included in the present study therefore had poorer self-efficacy in managing their disease, compared with the Put et al study, even though an improvement in self-efficacy was brought about by the intervention.

Overall, patients in the present study scored lower at baseline and post-intervention for the KASE-AQ compared with the patients included in the Put et al study. The questionnaires were orally administered in the patient’s primary language and every effort was made to ensure literacy level did not interfere with test administration, but the level of literacy of the sample population was very low, with many patients being unable to even write their own names. This could have lead to patients misunderstanding the questions or interpreting questions or words in an incorrect way.
4.4.6 SPIROMETRY MEASUREMENTS

4.4.6.1 %PEFR values

The following graph illustrates the baseline and post-intervention mean values for %PEFR in the Control and Intervention groups:

![Graph showing baseline and post-intervention mean values for %PEFR](image)

**Figure 4.35 Baseline and post-intervention mean values for %PEFR**

The Intervention Group showed an increase in %PEFR from 28.15% with a standard deviation of 17.57% at baseline, to a post-intervention %PEFR of 29.75% with a standard deviation of 15.01%. The Control Group also showed a small increase in %PEFR from 27.67% with a standard deviation of 15.19% at baseline to 28.57% with a standard deviation of 14.49% post-intervention. A p-value of 0.28574 was calculated using ANCOVA and this indicated that the improvement in %PEFR was not statistically significant since the p-value was greater than 0.05.

The study by Ngamviroj et al (2005)\(^79\) recorded a post-intervention morning %PEFR value of 65.77% with a standard deviation of 26.46%. This study by Ngamviroj et al therefore showed a slight decrease of 0.70% in morning %PEFR although the change was not significant (p=not significant). The values obtained for %PEFR in the present study were low compared with the study by Ngamviroj et al as well as other similar studies. Possible reasons for this are discussed below.
The South African Demographics and Health Survey 1998\textsuperscript{27} found that there was a sharp increase in the prevalence of abnormal PEFR with decreasing educational attainment. Men with no education showed a five times higher prevalence of abnormal PEFR than men with greater than Grade 12 education. The corresponding ratio for women was fourfold.\textsuperscript{27} Since the level of literacy in this study population was low it is possible that the reason for the low spirometry values could also be partly attributed to the lack of education in the study population. The influence of disability grants and the patients’ fear of losing their grants if their lung function improves also played a major role in the recording of such low spirometry values. It is also known that nearly 5\% of any population is incapable of performing an acceptable spirometry test, even after eight efforts.\textsuperscript{32}

A study by Apter \textit{et al} (1998)\textsuperscript{82}, which included fifty patients with moderate or severe asthma recruited from outpatient health centre-based clinics, also found that asthma control at the time of their study was somewhat poorer in individuals who were of minority status, had lower educational achievement, spoke Spanish as a primary language, were unemployed, or had a lower household income. Patients included in the present study were of a lower socio-economic status and were generally exposed to poorer living conditions. Their residential area was close to an industrial area and they were exposed to environmental pollutants on a daily basis. Childhood asthma was also possibly poorly managed and allergens in foodstuffs and from other sources might have played a role in the development of asthma in this sample population and the low spirometry values obtained in this study.

The South African Demographics and Health Survey 1998\textsuperscript{27} also showed a strong association between a history of diagnosed tuberculosis and airflow limitation, chronic bronchitis and abnormal PEFR among men and women.
No accurate statistics for the prevalence of pulmonary tuberculosis (PTB) at Kruisfontein Clinic could be found and PTB was not documented on patient clinic records but the following table indicates the incidence of PTB in South Africa:

Table 4.7 PTB statistics in South Africa as per Health Systems Trust website

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<th>EC</th>
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<td>42571</td>
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</tr>
</tbody>
</table>


From the above table it is evident that PTB is highly prevalent in the Eastern Cape and it can therefore also be assumed that a large number of patients included in this study may have suffered from PTB before. This could then also be a reason for the low spirometry values recorded during the course of the study.

A study by Urek et al (2004) found that PEFR was an especially good indicator of asthma activity and this suggestion has been reinforced by a number of other studies. However, this study showed that %FVC and %FEV₁ increased significantly (p<0.05) even though %PEFR did not increase significantly (p=0.29).
The following graph illustrates the baseline and post-intervention mean values for %FVC in the Control and Intervention groups:

![Graph showing baseline and post-intervention mean values for %FVC](image)

**Figure 4.36 Baseline and post-intervention mean values for %FVC**

* Indicates a statistically significant improvement (p<0.05)

The Intervention Group showed a significant increase in mean %FVC measurement from a mean %FVC of 44.80% with a standard deviation of 20.62% at baseline, to a post-intervention mean value of 48.35% with a standard deviation of 19.38% (p<0.05). The Control Group also showed a decrease in mean %FVC from 46.92% with a standard deviation of 16.81% to a post-intervention mean of 42.57% with a standard deviation of 16.78%. A p-value of 0.022 was calculated using ANCOVA and this indicated a significant effect on %FVC. The intervention therefore had a significant effect in improving %FVC in the Intervention Group at three months after randomisation. The adjusted mean was calculated to be 5.4691. Cohen’s $d$ was shown to be 0.3023 which indicated a small effect.

Kauppinen *et al* (1999)\(^80\) reported a post-intervention mean %FVC in the Intervention Group of 99.8%. The baseline mean %FVC in this study was 94.7%. The value obtained for post-intervention mean %FVC in the Intervention Group was therefore lower in the present study when compared with the Kauppinen *et al* study. The increase in mean %FVC in the Intervention Group was 3.55% in the present study when compared with a 4.9% increase in the Kauppinen *et al* study.
Illustrated below are the baseline and post-intervention mean values for %FEV$_1$ in the Control and Intervention groups:

![Graph showing mean %FEV$_1$ values for Control and Intervention groups before and after intervention.

Figure 4.37  Baseline and post-intervention mean values for %FEV$_1$

* Indicates a statistically significant improvement (p<0.05)

A significant increase in mean %FEV$_1$ measurement from a mean baseline %FEV$_1$ score of 43.33% with a standard deviation of 21.15% to a post-intervention mean of 47.73% with a standard deviation of 20.12% was recorded in the Intervention Group. The Control Group showed a small decrease in mean %FEV$_1$ from a baseline score of 43.27% with a standard deviation of 17.44% to a post-intervention score of 43.12% with a standard deviation of 17.35%. An adjusted mean increase of 4.7447 was also calculated.

ANCOVA yielded a p-value of 0.043 which indicated a significant increase in %FEV$_1$ in the Intervention Group since the p-value was less than 0.05. This intervention therefore had a significantly positive effect on mean %FEV$_1$ measurements in the Intervention Group. Cohen’s $d$ was calculated as 0.2531 which indicated a small effect.

Oga et al (2002)$^{81}$ produced a post-intervention mean %FEV$_1$ of 86.00% with a standard deviation of 17.90% in the Intervention Group of their study. The baseline %FEV$_1$ for this study was 71.60% with a standard deviation of 21.90%. This is lower
than the %FEV\textsubscript{1} values recorded for the present study. Possible reasons for this have been discussed under Section 4.3.6.1.

4.4.7 INHALER TECHNIQUE SCORES

The graph below illustrates the baseline and post-intervention mean scores for inhaler technique in the Control and Intervention groups:

![Bar chart showing inhaler technique scores](image)

**Figure 4.38 Baseline and post-intervention means for inhaler technique score**

* Indicates a statistically significant improvement (p<0.05)

The Intervention Group showed a significant increase in inhaler technique from a baseline mean inhaler technique score of 3.12 (maximum possible score of 7) with a standard deviation of 1.26, to a post-intervention mean score of 4.45 with a standard deviation of 1.24. An increase in inhaler technique score in the Control Group was also observed with a mean inhaler technique score of 2.75 with a standard deviation of 1.23 at baseline, which increased to a mean score of 3.02 with a standard deviation of 1.12 post-intervention. The adjusted mean increase was calculated as 1.2537.

Using ANCOVA, a p-value of 7.3x10\textsuperscript{-9} was calculated which indicated a statistically significant increase in inhaler technique in the Intervention Group. The improvement in inhaler technique in the Intervention Group was the most noticeable outcome of this study. Cohen’s \(d\) was calculated as 1.065 which indicated a large effect. A study by Cordina et al (2001)\textsuperscript{11} showed that 37.50% of patients in the Intervention Group in their study showed an improvement in inhaler technique post-intervention. An
intervention of this nature can therefore produce a significant improvement in inhaler technique which in turn can positively improve asthma management.

The following graph indicates the number of patients performing each of the seven specified steps used to assess inhaler technique correctly at baseline and post-intervention in the Control and Intervention groups:

![Graph showing number of patients performing correct actions](image)

**Figure 4.39** Baseline and post-intervention means indicating correct actions in the Control and Intervention groups for inhaler technique score

The most noticeable improvement in the Intervention Group was for step 7 which involved performing all six steps in the correct order. Patients also performed better for step 6 post-intervention. This involved waiting before the next inhalation and not trying to inhale more than one puff during one inhalation. An improvement was also seen with step 5 in the Intervention Group and the Control Group. This involved holding the breath after the inhalation for about ten seconds.

### 4.4.8 MEDICATION REGIMENS POST-INTERVENTION

Figure 4.40 indicates the total number of actual medication changes made. It does not take into account the patients in the Intervention Group who did not return or did not accept the prescribing changes or who did not return for the second interview. It was assumed that these patients’ medication regimens stayed the same.
The decrease in the amount of patients prescribed theophylline was the most noticeable change in medication regimens. At baseline 54 patients (90.00%) (n=60) in the Intervention Group were using theophylline tablets. This number decreased to 14 patients (23.33%) (n=60) post-intervention.

Prednisone usage decreased by almost 17% in the Intervention Group. Post-intervention only two patients (3.30%) in the Intervention Group were prescribed chronic systemic corticosteroids to manage their asthma condition. On the other hand, inhaled corticosteroid usage increased by 33.00%. A total of 30 patients (50.00%) were prescribed beclomethasone inhalation post-intervention. All of the abovementioned changes are in line with the guidelines.

Post-intervention patients in the Intervention Group were still using the salbutamol inhaler on its own, but used the spacer as recommended with the inhaled beclomethasone.
4.4 SUMMARY OF THE IMPACT OF THE INTERVENTION

The multi-faceted intervention had a significantly positive impact on several patient variables. Patients in the Intervention Group showed a significant increase in total quality of life (p=0.02332), as well as average quality of life (p=0.02369). This indicates that an intervention, including patient education on asthma, medication assessment and the suggestion of the use of medication aids such as a spacer, can significantly increase an asthmatic patient's overall quality of life. Although the increase in average quality of life score was statistically significant, it needs to be noted that it may not be clinically meaningful, since the increase was less than 0.5 on the seven point scale (see Section 4.3.4.2).

The activity limitation subscale of the quality of life questionnaire also showed a significant increase in score in the Intervention Group post-intervention (p=0.01049). This implied that, after education and medication assessment, asthmatic patients perceived that they could take part in more activities, for example playing with their children or doing household tasks, without suffering an asthma attack.

The patients’ asthma-related knowledge, as measured by the knowledge attitude and self-efficacy questionnaire (KASE-AQ), also increased significantly in the Intervention Group (p=0.037). Patients who had been educated on their disease state, showed a marked improvement in asthma-related knowledge post-intervention. Numerous other studies have also shown a marked improvement in asthma management and control with patient education.\textsuperscript{64-67} This therefore suggests that the increased knowledge of patients in this study group may contribute to an improvement in their asthma management and control.

With regards to lung function, patients in the Intervention Group showed a significant increase in mean values for %FVC (p=0.022) and %FEV\textsubscript{1} (p=0.043) post-intervention. %FVC is a measure of lung volume and %FEV\textsubscript{1} a measure of airflow. The ratio of FEV\textsubscript{1}/FVC can be seen as a highly sensitive measure of lung function.\textsuperscript{29} On the other hand however, %PEFR did not improve significantly. These
improvements in lung function can possibly be attributed to better inhaler technique, better medication regimens and spacer usage, as well as asthma education.

The most noticeable improvement post-intervention was the mean score for inhaler technique in the Intervention Group ($p=7.3\times10^{-9}$). Patients showed a marked improvement in inhaler technique after being educated on the correct use of a metered-dose inhaler. The most dramatic improvement was for step 6 and step 7 of the inhaler technique which involved waiting between puffs and performing all six steps in the correct order. This is of particular importance since only 10% to 30% of the metered dose usually gets deposited in the lung area with optimal technique.$^3$ Good inhaler technique is essential in ensuring that a therapeutically significant amount of the drug actually reaches the lungs. Patients were advised to use the customised spacer with the inhaled beclomethasone and therefore still required good inhaler technique for the salbutamol inhaler.

These results appear to be consistent with those of other similar studies. Chan et al (2004)$^7$ demonstrated that by improving PEFR and patient quality of life, a pharmacist intervention can be a cost-effective addition to the management of patients at an outpatient clinic. As was demonstrated in the present study, Chan et al also reported a significant improvement in asthma knowledge.$^7$ Kheir et al (2001)$^9$ showed that, with appropriate training and support, pharmacists can help asthma patients achieve greater quality of life.

In a systematic review of published randomised control trials assessing the effect of pharmaceutical care practice on patient outcomes, Roughead et al (2005)$^{10}$ found that pharmaceutical care services are effective in improving medication use although they do not provide sufficient evidence supporting improved health-related quality of life. However, in a study to assess the impact of a community-based pharmaceutical care program for patients with asthma, Cordina et al (2001)$^{11}$ reported a significant increase in quality of life. Kheir et al (2001)$^9$ also reported a significant increase in all subscales of the Asthma Quality of Life Questionnaire except for the environmental stimuli subscale.
A significant improvement in inhaler technique, as reported in the present study, was also documented by Cordina et al (2001)\textsuperscript{11}, who also used a categorical scoring system to assess inhaler technique.

The findings in the present study are therefore in line with and support the findings of other studies since a significant increase in total quality of life, average quality of life and activity limitations were reported.

Overall the multi-faceted pharmaceutical care intervention including medication assessment, asthma education and the recommendation of a medication aid in the form of a customised spacer produced significantly positive results in this population group.
CHAPTER 5
CONCLUSION AND RECOMMENDATIONS

5.1 CONCLUSION

In summary, post-intervention there was a statistically significant:

- improvement in total and average quality of life scores
- decrease in asthma-related activity limitations
- improvement in asthma-related knowledge
- improvement in lung function, as measured by %FVC and % FEV₁ and
- improvement in inhaler technique.

A highly significant improvement in inhaler technique was observed in the Intervention Group at the follow-up interview. Good inhaler technique is crucial in ensuring that a therapeutic amount of active drug actually reaches the lungs. The use of a customised 500ml plastic bottle as spacer, for use with the inhaled beclomethasone, was also recommended to 44 patients in the Intervention Group and, upon returning for reassessment, the patients displayed a positive attitude towards the use of a spacer.

The quality of life of patients as measured using the AQLQ(S) showed a statistically significant improvement post-intervention in mean total quality of life score and mean average quality of life score in the Intervention Group. This suggested that the patients’ overall quality of life regarding asthma therefore improved after the intervention.

The activity limitation subscale of the questionnaire showed an improvement in mean score in the Intervention Group post-intervention. This implied that following the pharmacist intervention, asthmatic patients could take part in more activities without suffering an asthma attack.
There was a significant improvement in asthma-related knowledge. The questionnaire used to measure knowledge, attitude and self-efficacy showed a significant improvement in mean knowledge score in the Intervention Group following the intervention. With regards to lung function measurements, which would be an indicator of asthma control, both %FVC and %FEV₁ improved significantly in the Intervention Group. Although not shown to be statistically significant, improvements in asthma-related attitude and self-efficacy, emotional function and %PEFR, as well as a decrease in symptoms and the impact of environmental stimuli, were also noted.

This study therefore showed that pharmaceutical care had a positive impact on the management and well-being of asthmatic patients. Improvements in lung function, asthma knowledge, attitudes and perceived self-management efficacy as well as asthma related quality of life and asthma control in patients at a primary health care level were brought about by this study.

The multi-faceted pharmaceutical care intervention, involving a change in prescribing regimens where necessary, as well as education on asthma, the necessity of medication compliance and awareness of good inhaler technique, resulted in improved asthma-related knowledge amongst patients. This increase in knowledge resulted in an improvement in inhaler technique and, together with the suggested use of a spacer for inhaled beclomethasone, may have contributed to the improvement in lung function observed. The positive impact on lung function may have resulted in the improvement in asthma-related quality of life as well as a reduction in activity limitation.

Pharmacotherapeutic interventions, with regards to medication changes and dosage changes, were accepted and implemented by the clinic doctor without any hesitation. This reinforces the importance of pharmacists’ role as part of the health care team and highlights the positive effects that teamwork from the health care professionals’ sides can have on patient management. The results of this study therefore contribute to the available body of knowledge relating to asthma control and management and suggest that further research in the field of pharmaceutical care with regards to asthma management in South Africa, are warranted. It also shows that pharmaceutical care can have a positive impact on the management of asthmatic patients in a primary health care setting.
5.2 LIMITATIONS

The following limitations apply to this research project:

○ A pilot study was not conducted and this could have lead to some problems and limitations in the study being identified and addressed to improve the validity of the data.

○ Patients were hesitant to use the spirometer correctly even after being educated on its use since many of them were receiving disability grants and feared that they might lose these grants if their conditions improved. Accurate assessment of lung function was therefore not possible. Patients’ asthma severity could therefore also not be determined adequately since the %PEFR values obtained using the spirometer were not necessarily a true reflection of lung function.

○ Not all patients included in the study returned to the clinic for the follow-up interviews and contact details for the patients were difficult to obtain since many of them did not have a telephone. Finding transport in bad weather when they could not walk to the clinic was also a problem for many patients.

○ The study is also limited by the use of only one clinic and one population group. This limits the generalisability of the study since assumptions on asthma in other population groups or residential areas can not be made.

○ The low level of literacy of the patients at the clinic also made education difficult, especially with regards to proper use of the inhalers and the use of the spirometer. Diagrams were used in an attempt to overcome this limitation. (See Appendix 6 and Appendix 8). Literacy levels were however not measured during the study and this could also have been a limitation to the study since the study design could have been adapted to suit the needs of a population with low literacy levels.
5.3 RECOMMENDATIONS

Arising from this study, the following recommendations are proposed:

*With regards to the ongoing care of asthmatic patients at the clinic:*  

- An educational intervention aimed at health care professionals (especially the nursing staff involved in prescribing) to encourage the appropriate adjustment of asthma medication where needed and to stress the importance of inhaled corticosteroid use as well as the side-effects associated with chronic systemic corticosteroid use, be implemented.

- A procedure for ensuring patient follow-up and return for medication and assessment be developed.

- Asthma education be provided to groups of patients at a primary health care (PHC) level to improve patient knowledge and attitude regarding their condition.

- The feasibility of including Beclate® 100µg inhaler in the EDL instead of Beclate® 50µg inhaler be investigated.

- The regular use of modified 500ml plastic bottles as spacers in the management of asthma patients be implemented.

*With regards to future research:*  

- The study should be extended to include an increased number of PHC clinics and asthma patients.

- Seek a suitable means of following up on patients, to ensure that they return for medication and assessment.
- Develop an alternative method of assessing asthma severity by analysing asthma medication needed to control the patient’s condition.

- Investigate further the feasibility of running a programme implementing the use of modified 500ml plastic bottles at PHC clinics and hospitals.

Notwithstanding the limitations of this study, it can be concluded that pharmacists can impact positively on the management and well-being of asthmatic patients. Multi-faceted pharmacist interventions, including asthma education, medication assessment, education on inhaler technique and the provision of medication aids in the form of spacers, can significantly improve an asthmatic patient’s condition. This is therefore an important focus area for future research which could include extending the project to other healthcare facilities throughout the Eastern Cape and the rest of South Africa.
REFERENCES


43. Talabere, L. The effects of an asthma education program on selected behaviors of school-age children who have recently experienced an acute asthma episode. 1990. The Ohio State University,Columbus, OH. (thesis)


therapeutic education programme for asthma patients: "un souffle nouveau". *Patient Education and Counseling*, 58,41-46.


Appendix 1

NMMU Human Ethics Committee Approval
Ref: N 01/11/03/07 [H06H-002][Provisional Approval]

Contact person: Mrs U Spies

29 March 2006

Ms Z Mostert
Faculty of Health Sciences
NMMU

Dear Ms Mostert

THE IMPACT OF PHARMACEUTICAL CARE SERVICES ON THE MANAGEMENT AND WELL-BEING OF ASTHMA PATIENTS IN A PRIMARY HEALTH CARE CLINIC

Your above-entitled re-application for ethics approval served at the March 2006 ordinary meeting of the Research Ethics Committee (Human).

The Committee APPROVED the application.

Please inform your co-investigators of the outcome. We wish you well with the project.

Yours sincerely

Prof R du Randt
Chairperson: Research Ethics Committee (Human)

cc: Department of Research Management
Faculty Officer, Faculty of Health Sciences
Appendix 2

Permission letter from Cacadu District Municipality
21 February 2006

Department of Pharmacy
P O Box 77000
Nelson Mandela Metropolitan University
PORT ELIZABETH
6031

Attention: Prof. B Potgieter
Ms Zhan Mostert

Dear Sir/Madam

PERMISSION TO CONDUCT RESEARCH

Your correspondence dated 2 February 2006, which is attached, refers.

Please note that at the Mayoral Committee of 22 February 2006 approval was
granted for the said research interviews to be conducted under the following
provisos which are to be adhered to:

a) that interviews conducted with staff and patients be on an informed and
   voluntary basis;
b) that the staff and patients identification be held confidential;
c) that the standard general student indemnity form be completed by the
   student researcher prior to the research being undertaken;
d) that the Department of Health advise the researcher of the above provisos

Attached is the general student further indemnity which is to be completed and
forwarded to the Manager: Health Services, Ms X Sandi of the Health
Department, Cacadu District Municipality.

Please note that the contact person to activate access to clinics is:

- Kouga Health Sub-district
ACPN Mrs. De Klerk at tel. (042) 295 1357 or Cell: 084 478 4290

Yours faithfully

MUNICIPAL MANAGER

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c-Drive/Vava/Typ06/MSWord/Health/Letters/P1
/Encl.
Appendix 3

Written explanation of project provided to study participants
Kontakpersoon: Mev. S. Burton

Geagte Pasiënt

U word versoek om deel te neem aan ‘n navorsingsprojek. Ons sal u van die nodige informasie voorsien om u te help om die doel van die studie te verstaan en aan u verduidelik wat van u as deelnemer verwag word. Hierdie riglyne sal insluit die risikos en voordele verbonde aan u deelname aan die projek sowel as u regte as deelnemer. U is welkom om die navorser te vra om enigeiets wat nie vir u duidelik is nie aan u te verduidelik.

Om deel te neem sal daar van u verwag word om geskrewe toestemming te verskaf wat sal insluit u handtekening en u voorletters sowel as die datum om te verseker dat u alles verstaan en die terme aanvaar.

U het die reg om u bekommernisse rakende die studie te eniger tyd bekend te maak. Meld asseblief enige probleme aangaande die studie om onmiddellik aan die navorser te verskaf. Die navorser se kontaknommers word verskaf en u is welkom om dit te skakel.

Dit is ook belangrik dat u bese dat die studie goedgekeur moet word deur die Navorsingsetiek Komitee (Menslik) van die universiteit. Die komitee bestaan uit ‘n groep onafhanklike kennis wat toesien dat die regte en welvaart van deelnemers aan navorsing verseker word en dat studies op ‘n etiese wyse uitgevoer word. Studies kan nie uitgevoer word sonder goedkeuring van die komitee nie. Navrae in verband met u regte as navorsingsdeelnemer kan gerig word aan die Navordsingsetiek Komitee (Menslik). U kan die direkteur kontak by Navorsingsbestuur (041) 504-4536.

As u nie daar geholpe raak nie, kan u skryf aan: Die Voorsitter van die Navorsing, Tegnologie en Innovasie Komitee, Posbus 77000, Nelson Mandela Metropolitaanse Universiteit, Port Elizabeth, 6031.

Deelname aan navorsing is heeltemal vrywillig. U word nie verplig om aan enige navorsing deel te neem nie. U huidige en/of toekomstige gesondheidsorg sal nie benadeel word as u verkies om nie aan medies-verwante navorsing deel te neem nie. U sal ook geen verlies aan voordele, wat u normaalweg op aanspraak het, ervaar nie.

As u wel deelneem kan u te eniger tyd onttrek, sonder verlies aan voordele en sonder diskriminasie teen u. U word versoek om asseblief terug te keer vir ‘n finale bespreking en ondersoek, om sodoende die navorsing op ‘n ordelike wyse af te sluit indien u sou onttrek van die studie.

U deelname mag beeindig word as u nalaat om instruksies te volg, weens administratiewe redes of as die navorser glo dat weens veranderinge aan u mediiese toestand dit tot u nadeel sal wees om voort te gaan met die studie.

Hoewel u identiteit te alle tye beskerm sal word, kan die resultate van die studie gebruik word by wetenskaplike konferensies of in spesialis publikasies.

Hierdie toestemmingsdokument is voorberei volgens huidige wetlike riglyne.

Die uwe

Zhan Mostert
082 264 6085
zmostert@hotmail.com
DIE NAVORSER
Appendix 4

Informed consent form
**INFORMASIE EN INGELIGTE TOESTEMMING VORM**

<table>
<thead>
<tr>
<th>Titel van projek</th>
<th>THE IMPACT OF PHARMACEUTICAL SERVICES ON THE MANAGEMENT OF ASTHMA PATIENTS AT A PRIMARY HEALTH CARE CLINIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Verwysingsnommer</td>
<td>Pasiënt nommer (studie): Leër nommer:</td>
</tr>
<tr>
<td>Hoofnavorser</td>
<td>Zhan Mostert</td>
</tr>
<tr>
<td>Adres</td>
<td>113 Elmdor</td>
</tr>
<tr>
<td>Postkodo</td>
<td>Port Elizabeth 6013</td>
</tr>
<tr>
<td>Kontak telefoonnommer</td>
<td>082 264 6085</td>
</tr>
</tbody>
</table>

**A. VERKLARING DEUR DEELNEMER**

<table>
<thead>
<tr>
<th>Voorletter</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<tr>
<td></td>
</tr>
</tbody>
</table>

**A.1 EK VERKLAAR HIERMEE DIE VOLGENDE:**

<p>| |</p>
<table>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
</tbody>
</table>

1. Ek, die deelnemer is uitgenooi om deel te neem aan die bogenoemde navoringsprojek wat onderneem word deur Zhan Mostert van die Departement van Aptekerswese in die Fakulteit van Gesondheidswetenskappe van die Nelson Mandela Metropolitaanse Universiteit.

2. **Die volgende aspekte is aan my, die deelnemer, verduidelik:**

2.1 **Doel:** Die navorser bestudeer: Die impak van farmaseutiese sorg-dienste op die behandeling van asma pasiënte in ’n primêre gesondheidssorg kliniek.

Die informasie sal gebruik word vir: “n Meesterprojek van die navorser sowel as vir die verbetering van die behandeling van asma pasiente by die genoemde kliniek.
## 2.2 Procedures
Ek verstaan die procedures soos verduidelik in die aangehegde brief.

## 2.3 Risikos
Geen eksperimentiële medikasie. Veranderinge sal aangebring word volgens die Nasionale Riglyne vir die Behandeling van Kroniese Asma in volwassesnes slegs na aanvaarding deur en onder supervisie van u mediese praktisyn. Naas die risikos normaalweg geassosieer met ’n verandering van medikasie (bv. neue-effekte) word geen ander risikos voorsien nie.

## 2.4 Moontlike voordele
As gevolg van my deelname aan hierdie studie kan my asma beheer of behandeling en daarom my asma-gebaseerde lewenskwaliteit verbeter.

## 2.5 Konfidensialiteit
My identiteit sal nie in enige besprekinge, verduideliking of wetenskaplike publikasies bekend gemaak word deur die navorser nie.

## 2.6 Beskikbaarheid van bevindinge
Enige nuwe informasie/of voordeel wat gedurende die verloop van die studie ontwikkel sal as volg bekend gemaak word: Dit sal ingehandig word in die vorm van ’n tesis wat beskikbaar sal wees in die NMMU biblioteek en mag gepubliseer word in geakkrediteerde joernale of aangebied word as ’n plakkaat of mondelinge voorlegging by ’n konferensie.

## 2.7 Vrywillige deelname/wyering/staking:

<table>
<thead>
<tr>
<th>My deelname is vrywillig</th>
<th>JA</th>
<th>NEE</th>
</tr>
</thead>
<tbody>
<tr>
<td>My besluit om deel te neem aan die studie of nie sal op geen wyse my huidige of toekomstige sorg/ leefstyl/ beroep beinvloed nie</td>
<td>WAAR</td>
<td>VALS</td>
</tr>
</tbody>
</table>

3. Die bogenoemde informasie is aan my verduidelik deur:

<table>
<thead>
<tr>
<th>Zhan Mostert</th>
</tr>
</thead>
</table>

in Afrikaans Engels

en ek is die bogenoemde taal ten volle magtig

Ek is die geleentheid gegun om vrae te vra en al my vrae is voldoende beantwoord.

4. Geen druk is op my uitgeoefen om my toestemming vir deelname te gee nie en ek verstaan dat ek te eniger tyd my deelname kan onttrek sonder enige benadeling.

5. Deelname aan hierdie studie sal nie lei tot enige addisionele uitgawes vir myself nie.
## A.2  EK GEE HIERMEE VRYWILLIGE TOESTEMMING OM AAN BOGENOEICDE PROJEK DEEL TE NEEM

<table>
<thead>
<tr>
<th>Geteken/bevestig by</th>
<th>op</th>
<th>20</th>
</tr>
</thead>
<tbody>
<tr>
<td>Handtekening van getuie</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Handtekening of regterduim afdruk van deelnemer</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Volle name van getuie</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

## B. VERKLARING DEUR NAVORSER

Ek, Zhan Mostert, verklaar dat:

- Ek die informasie verskaf in die dokument verduidelik het aan

- Hy/sy is aangemoedig om vrae te vra en voldoende tyd gegun hiervoor

- Hierdie gesprek het plaasgevind in Afrikaans en Engels en geen vertaler is gebruik gedurende die gesprek nie

- Ek het Afdeling C aan die deelnemer oorhandig

<table>
<thead>
<tr>
<th>Geteken/bevestig by</th>
<th>op</th>
<th>20</th>
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</thead>
<tbody>
<tr>
<td>Handtekening van onderhoudvoerder</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Volle name van getuie</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
C. BELANGRIKE BOODSKAP AAN DEELNEMER

Geagte deelnemer

Baie dankie vir u deelname aan die studie. Sou daar te eniger tyd gedurende die studie:
- ’n noodgeval ontstaan as gevolg van die studie, of
- u enige verdere inligting in verband met die studie benodig

Kontak asseblief by
Zhan Mostert
082 264 6085
Appendix 5

Patient data collection form
# Data collection form:

The impact of pharmaceutical care services on the management of asthma patients in a primary health care clinic

<table>
<thead>
<tr>
<th>Control Group</th>
<th>Intervention Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Consent</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Patient study number</th>
<th></th>
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</thead>
</table>

<table>
<thead>
<tr>
<th>File number</th>
<th>Sex</th>
<th>Male</th>
<th>Female</th>
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</thead>
</table>

<table>
<thead>
<tr>
<th>Age</th>
<th>yrs</th>
<th>Occupation</th>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Height</th>
<th>m</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Tel.number</th>
<th></th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Smoker?</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Weight</th>
<th>kg</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Home fuel use?</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Co-morbidities (acute/chronic)</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Current medication</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Repeat medication past 6mths</th>
</tr>
</thead>
<tbody>
<tr>
<td>6/6</td>
</tr>
<tr>
<td>5/6</td>
</tr>
<tr>
<td>4/6</td>
</tr>
<tr>
<td>3/6</td>
</tr>
<tr>
<td>2/6</td>
</tr>
<tr>
<td>1/6</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Six month medication history</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dates</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>NOTES</th>
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</thead>
</table>

156
### Approximate date of asthma diagnosis

<table>
<thead>
<tr>
<th>Date</th>
</tr>
</thead>
</table>

### Asthma symptom history (past six months)

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1/month</td>
<td>2-4/month</td>
</tr>
</tbody>
</table>

### Asthma medication history

<table>
<thead>
<tr>
<th>Dates</th>
</tr>
</thead>
</table>

### Asthma symptom severity (current)

#### Night-time symptoms

<table>
<thead>
<tr>
<th>Symptoms (wheeze, cough, tightness of chest &amp; waking)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1/month</td>
</tr>
</tbody>
</table>

#### Day-time symptoms

<table>
<thead>
<tr>
<th>Symptoms (cough, tight chest, wheeze)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;2/week</td>
</tr>
</tbody>
</table>

#### PEF

<table>
<thead>
<tr>
<th>PEF %</th>
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<tbody>
<tr>
<td>&gt;80%</td>
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#### MEASUREMENTS

<table>
<thead>
<tr>
<th>First appointment</th>
<th>Second appointment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Date</td>
<td>Date</td>
</tr>
</tbody>
</table>

### Quality of life questionnaire

<table>
<thead>
<tr>
<th>Category</th>
<th>Score</th>
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<tbody>
<tr>
<td>Total score</td>
<td></td>
</tr>
<tr>
<td>Symptoms</td>
<td></td>
</tr>
<tr>
<td>Activity limitation</td>
<td></td>
</tr>
<tr>
<td>Emotional function</td>
<td></td>
</tr>
<tr>
<td>Environmental stimuli</td>
<td></td>
</tr>
</tbody>
</table>

### Asthma knowledge questionnaire

<table>
<thead>
<tr>
<th>Score</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Total score for questionnaire</td>
<td></td>
</tr>
<tr>
<td>Score for knowledge (/20)</td>
<td></td>
</tr>
<tr>
<td>Score for attitude (/100)</td>
<td></td>
</tr>
<tr>
<td>Score for self-efficacy (/100)</td>
<td></td>
</tr>
</tbody>
</table>
### Education provided: (INTERVENTION GROUP ONLY)

<table>
<thead>
<tr>
<th>Comments:</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
</table>

### Proposed changes to medication (INTERVENTION GROUP ONLY)

<table>
<thead>
<tr>
<th>Accepted</th>
<th>Not accepted</th>
<th>Modified</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
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<td></td>
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</tbody>
</table>

**Comments:**

### Spirometry

<table>
<thead>
<tr>
<th>Peak expiratory flow rate</th>
<th>First appointment</th>
<th>Second appointment</th>
</tr>
</thead>
<tbody>
<tr>
<td>%PEFR</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FVC</td>
<td></td>
<td></td>
</tr>
<tr>
<td>%FVC</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FEV1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>%FEV1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Assessment of inhaler technique**

1. Remove cap & shake
2. Exhale slowly
3. Actuate at onset of inhalation
4. Inhale slowly
5. Hold breath
6. Wait before next inhalation
7. Correct sequence used

*(Tick block if action was correctly performed)*

**Use of bottle as spacer explained and suggested**

(INTERVENTION GROUP ONLY)

<table>
<thead>
<tr>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
</table>

### Education provided (INTERVENTION GROUP ONLY)

<table>
<thead>
<tr>
<th>Comments:</th>
<th></th>
<th></th>
<th></th>
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</table>

### Other comments:

<table>
<thead>
<tr>
<th>Comments:</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
</table>
Appendix 6

Modified Knowledge Attitude and Self-efficacy Questionnaire with pictures and Afrikaans translations used
The Knowledge, Attitude, and Self-Efficacy

Asthma Questionnaire

This survey contains a series of statements, written in the first person, concerning your opinions about your asthma. The survey also contains questions regarding your knowledge of asthma. Please read each of the items carefully; then, circle the letter that you feel answers the question best. Remember to CHOOSE ONLY ONE RESPONSE for each item. Thank you.

1. Following a healthy diet and lifestyle will help control my asthma.
   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

* For all questions with the above options as answers, the following translations were used:
   a. Waar
   b. Meesal waar
   c. Soms waar en soms vals
   d. Meesal vals
   e. Vals

2. Which one of the following is not a common asthma symptom?
   a. Sore, dry throat
   b. Coughing
   c. Chest tightness
   d. Wheezing
   e. Shortness of breath

   a. Seer, droë keel
   b. Hoes
   c. ‘n Toe bors
   d. ‘n Bors wat fluit
3. Which one of the following statements is true?

   a. Asthma can be the result of an emotional illness
   b. People bring asthma on themselves
   c. Asthma is the result of how children are raised
   d. Asthma is a physical illness
   e. Both A and D

   a. *Asma kan die gevolg wees van ‘n emosionele probleem*
   b. *Mense bring asma op hulself*
   c. *Asma is die gevolg van hoe kinders groot gemaak word*
   d. *Asma is ‘n fisieke probleem*
   e. *Beide A en D*

4. I can recognize the changes that occur in my lungs before an asthma attack begins.

   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

5. It is important for me to take my asthma medications as prescribed.

   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

6. Which one of the following is not a component of the respiratory system?

   a. Alveoli
   b. Larynx
   c. Trachea
   d. Bronchial tubes
   e. Duodenum

   a. *Alveoli*
   b. *Larynx*
   c. *Trachea*
   d. *Bronchiale buise*
   e. *Duodenum*
Pictures used for question 6

A.) Alveoli
B.) Larynx
C.) Trachea
D.) Bronchial Tubes
E.) Duodenum
7. The function of the lungs is to:
   a. Bring carbon dioxide in and push oxygen out
   b. Enhance cardiac output and increase stroke volume
   c. Bring oxygen in and push carbon dioxide out
   d. Cleanse the nasal passages and prevent ketoacidosis
   e. Bring oxygen in and push nitrogen out

   a. Om koolstofdioksied in te bring en suurstof uit te stoot
   b. Verbeter die hart se uitvoer en slagvolume
   c. Bring suurstof in en stoot koolstofdioksied uit
   d. Maak die neus se gange oop
   e. Bring suurstof in en stoot stikstof uit

8. I can do a great deal to solve the problems that asthma can cause.
   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

9. When it comes to my asthma, I feel that I can avoid having to miss work or other daily responsibilities.
   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

10. Oxygen is exchanged in the _____________:
    a. Larynx
    b. Alveoli
    c. Pancreas
    d. Bronchial tubes
    e. Trachea

    a. Larynx
    b. Alveoli
    c. Pankreas
    d. Bronchiale buise
    e. Trachea
Pictures used for question 10
11. I would like to learn as much as I can about how to manage my asthma.
   a. True  
   b. Mostly true  
   c. Sometimes true and sometimes false  
   d. Mostly false  
   e. False

12. Air needs to be ___________ before it reaches the lungs.
   a. Warmed  
   b. Humidified  
   c. Cooled  
   d. B and C  
   e. A and B
      a. Verwarm word  
      b. Klam gemaak word  
      c. Afgekoel word  
      d. B en C  
      e. A en B

13. I can prevent asthma in almost all situations.
   a. True  
   b. Mostly true  
   c. Sometimes true and sometimes false  
   d. Mostly false  
   e. False

14. My family can help me to remain calm during my asthma episodes.
   a. True  
   b. Mostly true  
   c. Sometimes true and sometimes false  
   d. Mostly false  
   e. False

15. I have confidence in my ability to keep my asthma under control when I am in a different city on vacation or on a business trip.
   a. True  
   b. Mostly true  
   c. Sometimes true and sometimes false  
   d. Mostly false  
   e. False
16. Which one of the following is not a common asthma trigger?
   a. Weather changes
   b. Laughing
   c. Aspirin
   d. Exercise
   e. Caffeine

   a. Weerveranderinge
   b. Lag
   c. Aspirien
   d. Oefening
   e. Kaffeïne

17. I can help my family remain calm during my asthma episodes.
   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

18. Which one of these physiological changes does not occur in the respiratory system before and during an asthma attack?
   a. The muscles around the bronchial tubes tighten
   b. The mucus in the bronchial tubes thickens
   c. The inner lining of the bronchial tubes swells
   d. The blood vessels of the bronchial tubes enlarge
   e. The airways narrow

   a. Die spiere rondom die bronchiale buise verstif
   b. Die slym in die bronchiale buise verdik
   c. Die binneste wand van die bronchiale buise swel op
   d. Die are rondom die bronchiale buise vergroot
   e. Die lugwëe vernou

19. I can take the necessary steps to avoid or to manage an asthma attack effectively.
   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

20. I feel comfortable taking my asthma medications when I am at work or away from home.
   a. True
   b. Mostly true
21. One in _______ people suffers from asthma:

   a. 20  
   b. 15  
   c. 10  
   d. 5   
   e. 3   

* The above questioned was used instead of a question regarding the incidence of asthma in the United States.

22. My asthma is not bad enough to warrant my having to learn asthma management strategies.

   a. True  
   b. Mostly true  
   c. Sometimes true and sometimes false  
   d. Mostly false  
   e. False

23. I feel confident in my ability to exercise without having an asthma attack.

   a. True  
   b. Mostly true  
   c. Sometimes true and sometimes false  
   d. Mostly false  
   e. False

24. Which one of the following statements is false?

   a. The best time to treat an attack is before it starts.  
   b. The longer you wait to treat an attack after it begins, the more likely the attack is to clear.  
   c. Modifying your activities, drinking clear liquids, and using your inhaler will help clear an attack.  
   d. An attack can be treated before it begins by paying attention to your medications, the environment, your asthma triggers, your early warning signs, and your health habits.  
   e. For some people, menstrual periods may trigger asthma attacks.

   a. Die beste tyd om ‘n asma aanval te behandel is voordat dit begin.  
   b. Hoe langer jy wag om ‘n asma aanval te behandel nadat dit begin het, hoe beter die kans dat die aanval sal opklaar.  
   c. ‘n Aanval kan opklaar deur jou aktiwiteite te verander, helder vloeistowwe te drink en jou pompie te gebruik
d. ‘n Aanval kan behandel word voordat dit begin deur jou medisyne reg te gebruik en jou omgewing, asma-snellers, vroëe waarskuwingstekens en gesondheidsgewoontes te beheer

e. Hul menstruele siklus kan ‘n asma-aanval veroorsaak in sommige mense.

25. My family can help me get the upper hand on my asthma.

   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

26. I do very well at perceiving the level of my asthma at all times, including when I am experiencing no asthma at all, when I am experiencing slight asthma, when I am experiencing moderate asthma, and when I am experiencing severe asthma.

   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

27. When I have an asthma attack and have no idea what caused it, I may have __________:

   a. Failed to take my asthma medications
   b. Unknowingly come into contact with one of my asthma triggers
   c. Been experiencing a great deal of stress lately
   d. Been unaware of or ignored my early warning signs
   e. All of the above

   a. Vergeet het om my asma medisyne te neem
   b. Onwetend met een van my asma-snellers in aanraking gekom het
   c. Onder baie stres was
   d. Onbewus was van of my vroëe waarskuwingstekens geignoreer het
   e. Al bogenoemde

28. My physician can handle my asthma without my having to become involved.

   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

29. I have confidence in my ability to keep my asthma under control when problems arise in my family.
30. Which one of the following may actually make an asthma attack worse?

a. Continuing to exercise or work once an attack begins
b. Resting instead of remaining active to clear the mucus
c. Pursed-lip breathing techniques
d. Drinking warm liquids
e. Using a bronchodilator during the attack

31. I feel as though I am well informed about my asthma.

a. True
b. Mostly true
c. Sometimes true and sometimes false
d. Mostly false
e. False

32. I can handle the problems that asthma may cause.

a. True
b. Mostly true
c. Sometimes true and sometimes false
d. Mostly false
e. False

33. The cause of exercise-induced asthma is ________________:

a. Cooling and drying of the airways
b. Overheating of the airways
c. Not taking in enough oxygen
d. Not being able to rid the lungs of carbon dioxide fast enough
e. Build-up of lactic acid

a. Afkoel en droog word van die lugwëe
b. Oorverhitting van die lugwëe
c. Nie genoeg suurstof in te neem nie
d. Nie vinnig genoeg van die koolstofdioksied ontslae kan raak nie
e. Ophou van melksuur
34. Three “Rs” that are helpful in treating an acute asthma attack are:

a. Readjust medications, Readjust food intake, and Readjust fluid intake
b. Rest, Relaxation, and Right breathing
c. Readjust medications, Restrict fluids, and Restrict eating
d. Record symptoms, Report to physician, and Refrain from drinking liquids
e. Record triggers, Remove all stressors, and Renew commitment to take medications on time

a. Verander medikasie, verander kosinname en verander vloeistofinname
b. Rus, ontspan en reg asemhaal
c. Verander medikasie, beperk vloeistof- en kosinname
d. Teken simptome aan, sien ‘n dokter en drink geen vloeistowwe
e. Teken asma-snellers aan, verwyder alle stress en neem jou medikasie op tyd

35. I can learn to be an effective asthma self-manager.

a. True
b. Mostly true
c. Sometimes true and sometimes false
d. Mostly false
e. False

36. If cigarette smoke is bothering me, I feel that I can ask the person to stop smoking.

a. True
b. Mostly true
c. Sometimes true and sometimes false
d. Mostly false
e. False

37. My life revolves around my asthma.

a. True
b. Mostly true
c. Sometimes true and sometimes false
d. Mostly false
e. False

38. To prevent asthma attacks, it is important to pay attention to ______________________:

a. My early warning signs and my asthma triggers
b. Good health habits and medication compliance
c. The environment
d. A and B
e. A, B, and C

a. My vroëe waarskuwingstekens en asma-snellers
b. **Goeie gesondheidsgewoontes en die neem van my medikasie op tyd**

c. **Die omgewing**

d. **A en B**

e. **A, B, en C**

39. The more I know about asthma, the more I can help myself.

   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

40. I resent my asthma because it limits my mobility.

   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

41. Two early warning signs of an impending asthma attack are:

   a. Emotional and attitude changes
   b. Physical changes and insomnia
   c. Physical and attitude/mood changes
   d. Dizziness and increased sweating
   e. Dysphoric mood and emotional changes

   a. **Emosionele en houdingsveranderinge**
   b. **Fisieke veranderinge en slaaploosheid**
   c. **Fisieke en houdingsveranderinge**
   d. ‘n Dronk kop en sweet
   e. ‘n Slegte bui en emosionele veranderinge

42. I feel that I can take my asthma medications as prescribed by my doctor.

   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

43. I feel that I have enough information about asthma to allow me to manage my asthma.

   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
44. I want to work in partnership with my physician in taking care of my asthma.
   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

45. Three “ABCs” that are helpful in treating an acute asthma attack are
   ____________________:
   a. Alleviate stress, Breathe rapidly, and Calm down
   b. Address maladaptive behaviors, Breathe in a shallow manner, and Cough frequently to clear mucus from lungs
   c. Adjust activities, use a Bronchodilator, and Consume clear, lukewarm liquids
   d. Ask for help, Blow into your peak flow meter, and Check your peak flow values

   a. Verminder stres, haal vinnig asem en kalmeer
   b. Verander slegte gewoontes, haal vlak asem en hoes gereeld om slym te verwyder
   c. Verander oefening, gebruik Asthavent® pompie en drink, helder louwarm vloeistowwe
   d. Vra vir hulp, gebruik jou lugvloeimeter en kontrolleer jou lugvloei lesings

46. During an asthma episode, I can refrain from panicking in order to better manage the attack.
   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

47. I have confidence in my ability to avoid frequent trips to the emergency room because of my asthma.
   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

48. Preventing asthma is a skill I can learn.
   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
d. Mostly false
e. False

49. Which one of the following instruments objectively measures lung functioning?

a. Sphygmomanometer
b. Peak flow meter
c. Glucometer
d. Stethoscope
e. Polygraph

a. Sphygmomanometer
b. Lugvloeimeter
c. Glukosemeter
d. Stetoskoop
e. Poligraaf
Pictures used for question 49

A.) Sphygmomanometer
B.) Peak flow meter
C.) Glucometer
D.) Stethoscope
E.) Polygraph
50. There is nothing I can do to relieve an asthma attack before it gets worse.
   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

51. I feel OK about asking for help during asthma attacks when I need to.
   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

52. I don’t have a lot of confidence in my ability to manage my asthma.
   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

53. I can generally figure out what is causing an episode of my asthma.
   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

54. Which one of the following indicates that your inhaler is empty?

   a. The inhaler stands up at the top of the water
   b. The inhaler lays flat on the bottom of the water
   c. The inhaler floats on its side on top of the water
   d. The inhaler floats on a diagonal toward the top of the water
   e. The inhaler stands up on the bottom of the water

   a. Die pompie staan regop aan die bokant van die water
   b. Die pompie lê plat op die bodem van die water
   c. Die pompie dryf op sy kant aan die bokant van die water
   d. Die pompie dryf skuins aan die bokant van die water
   e. Die pompie staan regop op die bodem
55. Once an attack starts, I am not capable of stopping it; I just have to wait until it subsides.

   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

56. I want to take an active role in managing my asthma.

   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

57. I have a lot of confidence in my ability to detect the early warning signs of my asthma

   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False

58. Possible side effects of theophylline (Nuelin®) may include:

   a. Visual disturbances, sweating, and confusion
   b. Memory disturbances, increased appetite, and water retention
   c. Insomnia, weight gain, and depressed mood
   d. Vomiting, headache, and irritability
   e. Fatigue, restlessness, and slurred speech

   a. Visuele probleme, sweet en verwarring
   b. Geheueveranderinge, ‘n eetlus en waterretensie
   c. Slaaploosheid, gewigstoename en depressie
   d. Naarheid, kopseer en geïrriteertheid
   e. Moegheid, rusteloosheid en ‘n sleeptong

59. I can avoid or minimize most of my asthma triggers.

   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False
60. I can use positive self-talk to help control my asthma.

   a. True
   b. Mostly true
   c. Sometimes true and sometimes false
   d. Mostly false
   e. False
Appendix 7

Asthma Quality of Life Questionnaire
Short Form used in Afrikaans
ASMA LEWENSKWALITEIT VRAELYS
MET GESTANDARDEERDE
AKTIWITEITE (AQLQ(S))

ONDERVRAER-GEADMINISTREER
(INTERVIEWER-ADMINISTERED)
AFRIKAANS VERSION

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QOL TECHNOLOGIES LTD.

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This translation has been made possible
through a grant from BYK GULDEN
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Nadruk van die AQLQ(S) is verbode. Dit mag nie verander, verkoop (papier
of elektronies), vertaal of van 'n ander medium aangepas word sonder die
toestemming van Elizabeth Juniper nie.

MEI 2002
INLEIDING

DIE ASMA LEWENSKWALITEIT VRAELYS IS GETOEETS EN BEKRAGTIG MET DIE GEBRUIK VAN DIE BEWOERING EN FORMAAT WAT VOLG. DIT IS BELANGRIK DAT ONDERVRAERS BY DIE PRESIESE BEWOERING BLY WANNEER DIE PASiëNT AANGESPREEK WORD (GEWONE DRUK) EN DIE INSTRUKSIES VOLG (SKUINSDRUK). AFWYKING VAN SOWEL BEWOERING AS INSTRUKSIES MAG DIE BETROUBAARHEID EN GELDIGHEID VAN DIE VRAELYS BELEMMER.

DIE VRAELYS

Hierdie vraelys is ontwerp om uit te vind hoe u gedurende die afgelope twee weke gevoel het. U sal gevra word oor maniere waarop u asma u aktiwiteit beperk het, die simptome wat u ondervind het as gevolg van u asma, en hoe hierdie u laat voel het.

OORHANDIG DIE ANTWOORDVEL AAN DIE PASiëNT. VERDUIDElik DAT U WIL HE DAT DIE PASiëNT ELKE VRAAG BEANTWOORD DEUR DIE NOMMER VAN DIE ANTWOORD IN DIE TOEPASLIKE Ry EN KOLOM IN TE SKRYF. BY DIE EERSTE BESOEK SAL DIE ANTWOORDE IN DIE EERSTE KOLOM WEES.

BY ELKE OPVOLGBESOEK WORD PASiëNTe GELAS OM KENNIS TE NEEM VAN DIE TELLING WAT HULLE BY HUL VORIGE BESOEK INGESKRYF HET.

VOORDAT ELKE VRAAG GELEES WORD, MAAK SÉKER DAT DIE PASiëNT NA DIE REGTE GEKLEURDE ANTwoordKAART KYK.

Hoe beperk was u gedurende die afgelope 2 weke in hierdie aktiwiteite as gevolg van u asma?

A 1. Dui asseblief aan hoeveel u gedurende die afgelope 2 weke in VEELEISENDE AKTIWITEITE (soos haastig wees, oefening doen, teen trappe ophardloop, sport) deur u asma beperk is. [GROEN KAART]

A 2. Dui asseblief aan hoeveel u gedurende die afgelope 2 weke in MATIGE AKTIWITEITE (soos stap, huiswerk, tuinwerk, inkopies doen, trappe klim) deur u asma beperk is. [GROEN KAART]
3. Dui asseblief aan hoeveel u gedurende die afgelope 2 weke in **SOSIALE AKTIWITEITE** (soos praat, met troeteldiere/kinders speel, vriende/familie besoek) deur u asma beperk is. [GROEN KAART]

4. Dui asseblief aan hoeveel u gedurende die afgelope 2 weke in **WERKVERWANTE AKTIWITEITE** (take wat u by die werk moet doen*) deur u asma beperk is. [GROEN KAART]

*As u nie in diens is of vir uself werk nie, moet dit take wees wat u die op meeste dee moet doen.

5. Dui asseblief aan hoeveel u gedurende die afgelope 2 weke in u **SLAAP** deur u asma beperk is. [GROEN KAART]

6. Hoeveel ongemak of benoudheid het u oor die afgelope twee weke gevoel as gevolg van 'n **TOE BORS**? [ROOI KAART]

7. In die algemeen, hoe dikwels gedurende die afgelope twee weke het u **BESORG GEVOEL OMDAT U ASMA HET**? [BLOU KAART]

8. Hoe dikwels gedurende die afgelope twee weke het u **KORTASEM** gevoel as gevolg van u asma? [BLOU KAART]

9. Hoe dikwels gedurende die afgelope twee weke het u asma-simptome ondervind as gevolg van **BLOOTSTELLING AAN SIGARETROOK**? [BLOU KAART]

10. Hoe dikwels gedurende die afgelope twee weke het u 'n **GEFLUIT** in u bors ondervind? [BLOU KAART]

11. Hoe dikwels gedurende die afgelope twee weke het u gevoel dat u 'n **SITUASIE OF OMGEWING moes VERMY AS GEVOLG VAN SIGARETROOK**? [BLOU KAART]

12. Hoeveel ongemak of benoudheid het u oor die afgelope twee weke gevoel as gevolg van **GEHOES**? [ROOI KAART]
In die algemeen, hoe dikwels gedurende die afgelope twee weke het u GEFRUSTREERD gevoel as gevolg van u asma? [BLOU KAART]

Hoe dikwels gedurende die afgelope twee weke het u 'n DRUK GEVOEL OP DIE BORS ondervind? [BLOU KAART]

Hoe dikwels gedurende die afgelope twee weke het u BESORGE GEVOEL OOR DIE NODIGHEID OM MEDIKASIE TE GEBRUIK vir u asma? [BLOU KAART]

Hoe dikwels gedurende die afgelope twee weke het u die behoefte gehad om KEEL SKOON TE MAAK? [BLOU KAART]

Hoe dikwels gedurende die afgelope twee weke het u asma-simptome ondervind as gevolg van BLOOTSTELLING AAN STOF? [BLOU KAART]

Hoe dikwels gedurende die afgelope twee weke het u MOEITE MET UIT- OF INASEM ondervind as gevolg van u asma? [BLOU KAART]

Hoe dikwels gedurende die afgelope twee weke het u gevoel dat u 'n SITUASIE OF OMGEWING MOES VERMY AS GEVOLG VAN STOF? [BLOU KAART]

Hoe dikwels gedurende die afgelope twee weke het u IN DIE OGGEND WAKKER GEWORD MET ASMA-SIMPTOME? [BLOU KAART]

Hoe dikwels gedurende die afgelope twee weke het u BANG GEVOEL OM NIE U ASMA MEDIKASIE BESIKKBaar TE Hê NIE? [BLOU KAART]

Hoe dikwels gedurende die afgelope twee weke is u gepla deur SWAAR ASEMHALING? [BLOU KAART]

Hoe dikwels gedurende die afgelope twee weke het u asma-simptome ondervind as gevolg van die WEER OF LUGESEODELING Buite? [BLOU KAART]
24. Hoe dikwels gedurende die afgelope twee weke is u IN DIE NAG WAKKER GEMAAK deur u asma? [BLOU KAART]

25. Hoe dikwels gedurende die afgelope twee weke moes u VERMY OF USELF BEPERK OM Buite toe te gaan as gevolg van DIE WEER OF LUGBESOEDELING? [BLOU KAART]

26. Hoe dikwels gedurende die afgelope twee weke het u asma-simptome ondervind as gevolg van BLOOTSTELLING AAN STERK REUKE OF PARFUUM? [BLOU KAART]

27. Hoe dikwels gedurende die afgelope twee weke het u BANG GEVOEL OM UITASEM TE RAAK? [BLOU KAART]

28. Hoe dikwels gedurende die afgelope twee weke het u gevoel dat u 'n SITUASIE OF OMGEWING MOES VERMY AS GEVOLG VAN STERK REUKE OF PARFUUM? [BLOU KAART]

29. Hoe dikwels gedurende die afgelope twee weke het u asma INGEMENG MET 'N GOEIE NAGRUS? [BLOU KAART]

30. Hoe dikwels gedurende die afgelope twee weke het u die gevoel gehad van SNAK NA ASEM? [BLOU KAART]

31. Dink aan AL DIE VERSKILLENDE AKTIWITEITE wat u gedurende die afgelope twee weke sou wou doen. Hoeveel was u reeks van aktiwiteit deur u asma beperk? [GEEL KAART]

32. In die geheel, onder AL DIE AKTIWITEITE wat u gedoen het gedurende die afgelope twee weke, hoe beperk was u deur u asma? [GROEN KAART]

**GEBIEDSKODE:**

| S  | Simptome |
| A  | Aktiwiteitsbeperking |
| EM | Emosionele funksionering |
| EN | Omgewingsprikkels |
ANTWOORD OPSIES

GROEN KAART. Vrae 1 - 5 en 32
1.    TOTAAL BEPERK, KON GLAD NIE AKTIVITEIT DOEN NIE
2.    UITERS BEPERK
3.    BAIE BEPERK
4.    REDELIK BEPERK
5.    MIN BEPERK
6.    BAIE MIN BEPERK
7.    GLAD NIE BEPERK NIE

ROOIE KAART. Vrae 6 en 12
1.    'N BAIE GROOT MATE VAN ONGEMAK OF BENOODHEID
2.    'N GROOT MATE VAN ONGEMAK OF BENOODHEID
3.    HEELWAT ONGEMAK OF BENOODHEID
4.    'N MATIGE HOEVEELHEID VAN ONGEMAK OF BENOODHEID
5.    MIN ONGEMAK OF BENOODHEID
6.    BAIE MIN ONGEMAK OF BENOODHEID
7.    GEEN ONGEMAK OF BENOODHEID
BLOU KAART. Vrae 7 - 11 en 13 - 30
1. ALTYD
2. MEESTAL
3. NOGAL DIKWELS
4. SOMTYDS
5. NOU EN DAN
6. BYNA NOOIT
7. NOOIT

GEEL KAART. Vraag 31
1. ERG BEPERK - DIE MEESTE AKTWITEITE NIE GEDoen NIE
2. BAIE BEPERK
3. REDELIK BEPERK - VERSKEIE AKTWITEITE NIE GEDoen NIE
4. EFFENS BEPERK
5. BAIE EFFENS BEPERK - BAIE MIN AKTWITEITE NIE GEDoen NIE
6. FEITLIK GLAD NIE BEPERK NIE
7. GLAD NIE BEPERK NIE - HET AL (DIE) AKTWITEITE GEDoen WAT EK WOU DOEN
Appendix 8

Educational material used
Asma: Wat jy moet weet

Wat geheur in my longe?
Asma veroorsaak dit die kante van die lugweg in jou longe geuer is die hees, die longe raak in as jy suur, stof en stofmeel en die lugweg raak dan dus en begin skree wat die asma-symptome veroorsaak.

Simptome van asma
• Jy voel uitsasem
• ’n Bors wat fluut
• ’n Toe bors
• Hoës
Hierdie simptome is gewoonlik erger in die aard en vroeër in die opgang

Wat veroorsaak asma?
• Dinge wat jou allergies maak soos honde, katte, kokarolle, stofmeel
• Sommige pills soos Dispen®, Bruvans® en Inzali®
• Infeksies in jou longe
• Sommige kos wat jou eet
• Rook
• Detergens
• Koue weer

Wat beteken dit dat ek nou asma het?
• Asma wat nie goed beheer word nie kan baie probleme veroorsaak. Dit kan veroorsaak dat jy uit die werk bly, in die hospitaal moet lig of selfs doodgaan.
• Jy hoef nie te sukkel met asma nie. Daar is goeie medikasie wat jou simptome kan beter maak.

Jou asma medisyne
Daar is twee soorte medisyne vir asma:

1. Kontrolers: die wat langtermyn asma simptome verhoed soos jou bruin Beclate® pompie
2. Verlighers: die wat korttermyn verlig jou simptome kan verbeter soos jou blou Asthavent® pompie
1) Kontrolers

- Jou Nuelin® tablette en jou bruin Becate® pompje behoort aan hierdie groep.
- Hulle keer dat jou asma simptome verskyn.
- Dit is baie belangrik dat jy hierdie medisynge gereed sal gebruik, soos jy moet, selfs al voel jy nie asof jy dit nodig het nie. Dit sal keer dat jy asma erger word.

2) Verligters

- Jou blou Asthavent® pompje behoort aan hierdie groep.
- Dit word gebruik vir 'n asma- aanval en maak jou bors jou oop.
- Dit is belangrik dat jy jou Asthavent® pompje reg gebruik as jy dit regdig nodig het om net jou longe later gewoon te maak aan hom as hy hom te veel gebruik. Dan gaan hy nie meer so goed werk nie.

Nuelin® tablette

- Here die pille help keer dat jou bors toetrek.
- Dit is belangrik dat jy na die kliniek of hospitaal sal gaan as jy hertklopings kry, meer bewe as gewoonlik of meer koppeer kry as gewoonlik tervel jy hierdie pille drink.
- Moet nie rook of drink as jy die pille drink nie.

Jou Becate® pompje

- Here die pompje keer dat jou asma simptome verskyn.
- Jy moet die pompje gebruik selfs al het jy nie 'n toe-bors nie. Dit keer dat jou bors toetrek.
- Spoel jou mond uit nadat jy die pompje gebruik het. Dit keer dat jy sproei kry in jou mond.

Jou Asthavent® pompje

- Here die pompje is slegs om te gebruik as jy asma simptome soos toetrek of hertklopings kry. Gebruik jou Asthavent® pompje slegs as jy voel dat jou bors toe is, of as jy hoë of kortlauwe voel.
- Moet nie die pompje meer gebruik as dit nie, want later gaan jou bors nie meer opgaan nie as jy hom gebruik nie.
- Jy kan beweag voel nadat jy die pompje gebruik het.
- Jou Asthavent® pompje kan nie keer dat jou asma simptome verskyn nie. Want die Kontrolers, soos jou Becate® pompje en Nuelin® tablette kan dit doen.

Hoe om jou pompies reg te gebruik

- Dit is baie belangrik dat jy jou pompies reg gebruik, anders gaan jou medisyn nie werk soos dit moet nie.
- As dit vir jou beter is, gebruik die 500ml plastisubat van jou gevysis. Dit maak dit makliker om jou pompies reg te gebruik en keer dat jy sproei in jou mond kry.
Hoe om jou pompie reg te gebruik
1. Haal die dopje af en skud die pompie goed.
2. Blows al jou asma uit.
3. Dit die monstuk van die pompie in jou mond.
4. Sosé by stadiê deur jou mond begin insien.
5. Druk die pompie een keer en hou aan in as em so diep as wat jy kan.
6. Hou jou asma op toeloat jy tot die tien getel het.
7. Wág ten minste dertig sekondes voordat jy die stappe hê al vir die tweede puff.

Hoe om jou asma te beheer
1) Werk saam met die dokter en vanperskragters by die kliniek en doen wat hulle vir jou sé.
2) Neem jou medisyne presies soos jy gesê is om dit te neem.
3) Kyk uit vir simptome wat wys dat jou asma baie beheer raak en soek dadelik hulp.
4) Bly weg van dinge wat jou asma erger maak.

Dinge wat jou asma kan erger maak
• Jy moet vir jouself uitvind wat jou asma erger maak.
• Dinge wat by sommige mense asma vererger, is mischien glad nie 'n probleem vir jou asma nie en anders om.
• Verwyder eerste die dinge in jou slaapkamer wat jou asma erger maak.
• Hier onder is 'n lys van dinge wat almal 'n probleem kan wees en hou jy dit kan keer.

Dinge wat jou asma kan erger maak
• Sigarettensmoor- Probeer jou bes aan op te hou rook en vra ander mense om nie naby jou te rook nie.
• Miel- Was jou beddegoed elke week in baie warm water.
• Stofstof- Bly uit die kamer as daar gestoof is.
• Stofmeel- Hou jou vensters toe.