The Short Term Relative Effectiveness of Two Manual Interventions in the Management of Chronic Moderate Asthma

By

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I, Shekaar Ramesh Rampersad, do declare that this dissertation is representative of my own work in both conception and execution (except where acknowledgements indicate to the contrary)

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DEDICATION

This dissertation is humbly dedicated to God Almighty Lord Krishna for blessing me with a wonderful family:

My dad Ramesh, my mum Madhu, my sister Vashna and My rani Natasha

Without their unwaivering love and support throughout my life this all would not have been possible. I could never repay you all back for your kindness, patience, generosity and love. I love you all. May every child be blessed with a family like mine.

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ABSTRACT

Objectives:

- To determine the short-term effect of an inhaled, short-acting β_2-agonist bronchodilator on chest wall expansion (cm) and lung function parameters (FEV_1, FVC and FEV_1/FVC%) in chronic moderate asthmatics.
- To determine the short-term effect of spinal manipulation (SMT) and ribcage mobilisation on chest wall expansion (cm) and lung function parameters (FEV_1, FVC and FEV_1/FVC%) in chronic moderate asthmatics.
- To determine the short-term effect of a combination of SMT, ribcage mobilisation and an inhaled, short-acting β_2-agonist bronchodilator on chest wall expansion (cm) and lung function parameters (FEV_1, FVC and FEV_1/FVC%) in chronic moderate asthmatics.

Methods:

Forty-five chronic moderate asthmatics who met all the inclusion criteria of the study were divided into three groups of fifteen each. Group A received a short-acting β_2-agonist bronchodilator, Group B received SMT and ribcage mobilisation and Group C received a combination of SMT, ribcage mobilisation and a short-acting β_2-agonist bronchodilator. Baseline measurements and testing included chest wall expansion and the lung function parameters FEV_1, FVC and FEV_1/FVC%. These measurements were repeated 15 minutes post-intervention. Data was analyzed using SPSS version 15.0.

Results:

There were no statistically significant changes between pre- and post-intervention in the short-acting β_2-agonist bronchodilator group with respect to any of the chest wall expansion measurements. There was a statisticallly significant increase in FEV_1 between pre- and post-intervention in the short-acting β_2-agonist bronchodilator group (p = 0.008). There was a statistically significant increase in the mean pre- and post-intervention axillary chest wall expansion (p = 0.014) as well as the mean of the half-way measurement (p = 0.014) and the overall mean chest wall expansion value (p = 0.001) following SMT and ribcage mobilisation.

There were no statistically significant changes in any of the lung function parameter values following SMT and ribcage mobilisation. There was a significant increase for the half-way measurement in chest wall expansion (p = 0.018) in the combination of SMT, ribcage mobilisation and the inhaled, short-acting β_2-agonist bronchodilator group. There were no statistically significant changes in any of the lung function parameter values in the combination of SMT, ribcage mobilisation and an inhaled, short-acting β_2-agonist bronchodilator. For FEV_1, the effect in the short-acting β_2-agonist bronchodilator group *vs*. the SMT and ribcage mobilisation group was statistically significant (p = 0.018). There was no statistical difference in any of the chest wall expansion measurements and FVC and FEV_1/FVC% parameters between all three groups.

Conclusions

The results did not point specifically to one intervention over another for all outcomes. SMT and rib mobilisation had no effect on the lung function parameters, at least in the short term. There was a statisticallly significant increase in FEV_1 between pre- and post-intervention in the short-acting β_2-agonist bronchodilator group

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LIST OF DEFINITIONS

Short-term: For the purposes of this study “short-term” was defined as a time period of 15 minutes as bronchodilation occurs within five to 15 minutes after inhalation of a short-acting β_2 agonist bronchodilator (Gibbon, 2003).

Two manual interventions: The two manual interventions utilized in this study were spinal manipulation of the thoracic spine and ribcage mobilisation.

LIST OF ABBREVIATIONS AND SYMBOLS

ABG:	Arterial blood gas
AP:	Anteroposterior
$\beta_{2:}$	Beta two
BMI:	Body mass index
CAM:	Complementary and alternative medicine
cm:	Centimetres
C0 – C2:	Occiput to cervical vertebra 2
ed:	Edition
Ed:	Editor
EIA:	Exercise induced asthma
FEF_{25}:	Forced expiratory flow after 25% of expired
FEV_1:	Forced expiratory volume in one second
FEV_1/FVC%:	Percentage of the FVC
FVC:	Forced vital capacity
FVC; FEF_{75}:	Forced expiratory flow after 75% of expired
FVC; $FEF_{25\text{-}75.75}$:	Mean expiratory flow during the middle half of the FVC
GTOs:	Golgi tendon organs
hr:	Hour
kg:	Kilogram
IAPs:	Inferior articular processes
IgA:	Immunoglobulin A
IgE:	Immunoglobulin E
IU:	International units
L:	Litres
L/minute:	Litres per minute
L1-L5:	Lumbar vertebra with corresponding number
LT-C4:	Leukotriene complement 4

m^2:	Square meters
mcg:	Micrograms
mg:	Milligram

:

mg/kg/day:	Milligrams per kilogram per day
min:	Minutes
ml:	Millilitres
MMFR:	Maximal mid-expiratory flow rate
mm hg:	Millimetres mercury
MMV:	Maximum voluntary ventilation
Mnts:	Months
Mth:	Month
***n*:**	Sample size
N/A:	Not applicable
NO:	Nitric oxide
PEF:	Peak expiratory flow
pts:	Patients
RCTs:	Randomised clinical trials
ROM:	Range of motion
Rx:	Treatment
Rxs:	Treatments
SD:	Standard deviation
SMT:	Spinal manipulative therapy
SP:	Spinous process
STT:	Soft tissue therapy
T1 – T12:	Thoracic vertebra with corresponding number
TVPs:	Transverse processes
viz.:	Namely
vs.:	Versus
WHO:	World Health Organization
wk:	Week
wks:	Weeks
↓:	Decrease/reduction
>:	Greater than.
≥:	Greater than or equal to
↑:	Increase/improvement

<:	Less than
®:	Registered trademark

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CHAPTER ONE

INTRODUCTION

1.1. INTRODUCTION TO THE STUDY

Asthma, a pulmonary condition that is usually allergic in origin, is characterized by reversible airflow obstruction and airway inflammation, persistent airway hyperreactivity and airway remodelling (Morris and Perkins, 2005; Corrigan, 2008). Chronic asthma is classified into three categories depending on the person's symptoms and peak expiratory flow (PEF) readings viz. mild, moderate and severe asthma.

Forced expiratory volume in one second (FEV_1) is the volume of air forcefully expired during the first second after a full breath (Johns and Pierce, 2007), while forced vital capacity (FVC) is the volume of air expired with maximal force (Ganong, 1995). The FEV_1/FVC% provides a clinically useful index of airflow limitation (Ganong, 1995; Johns and Pierce, 2007). Spirometry, which primarily measures the FEV_1 and the FVC, is the recommended method of measuring airflow limitation and reversibility in order to establish a diagnosis of asthma (Haslett *et al.,* 2003; Morris and Perkins, 2005; Camargo, 2006; Gina Report, 2007). In asthma the FEV_1 is usually decreased, the FVC is usually normal and the ratio FEV_1/FVC% is decreased (Gina Report, 2007; National Asthma Council of Australia, 2008).

Asthma is managed primarily by the use of pharmacological agents where the intervention is aimed at reducing bronchial hyperresponsiveness, inflammation and bronchospasm. Medications to treat asthma can be classified as controllers or relievers (Rabe and Schmidt, 2001; Gibbon, 2003; Gina Report, 2007). Controller medications (e.g. corticosteroids) are taken in order to achieve clinical control of asthma primarily through their anti inflammatory effects (Risenga *et al.,* 2002; Gibbon, 2003; Gina Report, 2007) while reliever medication (e.g. inhaled, short-acting β_2-agonist bronchodilators) act quickly to relieve bronchoconstriction and its accompanying acute symptoms (Gibbon, 2003; Gina Report, 2007).

Recently, there has been growing interest shown in the use of non-pharmacological interventions as adjuncts to drug therapy in the management of asthma (Eisenberg *et al.*, 1993). These

interventions include acupuncture, yoga, hypnotherapy and manual therapies such as spinal manipulation (SMT). SMT, which involves the use of a short lever, high-velocity thrust of controlled amplitude on spinal articulations, often results in improved flexibility and increased joint mobility (Gal *et al*., 1994; Herzog *et al*., 1988; Herzog, 2000; Gatterman *et al*., 2001; Gatterman, 2003; Sood, 2008). According to Jamison *et al*. (1986), several practitioners of SMT were of the opinion that SMT has a role in the management of asthmatic patients. While there has been anecdotal and research evidence (Gobrin, 1997; Nilsson and Christiansen, 1998; Bronfort *et al.*, 2001; Leboeuf-Yde *et al.*, 2005) to suggest that SMT may be beneficial as an adjunct to regular pharmacological treatment in chronic asthmatics, some studies (Nielsen *et al*., 1995; Jamison *et al*., 1996; Balon *et al*., 1998; Hondras *et al.*, 2000) have, however, suggested that SMT may have no benefit as an adjunct to pharmacological intervention in chronic asthmatics.

Triano (2005) defined joint mobilisation as “cyclic application of joint loads at lower velocity” which is administered by a clinician for the purposes of increasing overall range of motion (ROM). A study by Kriel (2005) has shown that rib mobilisation resulted in an increase in chest wall expansion and lung function in healthy, asymptomatic males compared to a placebo and a control group. He postulated that the addition of ribcage mobilisation to another manual therapy may be beneficial as an adjunctive therapy in the management of asthma.

1.2. AIMS AND OBJECTIVES OF THE STUDY

The primary aim was to determine the short-term effect of:

- An inhaled, short-acting β_2-agonist bronchodilator,
- SMT and ribcage mobilisation and
- Combination of SMT, ribcage mobilisation and an inhaled, short-acting β_2-agonist bronchodilator

on chest wall expansion and lung function in chronic moderate asthmatics.

Several specific objectives were identified and these included:

1.2.1 To determine the short-term effect of an inhaled, short-acting β_2-agonist bronchodilator on chest wall expansion (cm) and lung function parameters (FEV_1, FVC and FEV_1/FVC%) in chronic moderate asthmatics.

1.2.2 To determine the short-term effect of SMT and ribcage mobilisation on chest wall expansion (cm) and lung function parameters (FEV_1, FVC and FEV_1/FVC%) in chronic moderate asthmatics.

1.2.3 To determine the short-term effect of a combination of SMT, ribcage mobilisation and an inhaled, short-acting β_2-agonist bronchodilator on chest wall expansion (cm) and lung function parameters (FEV_1, FVC and FEV_1/FVC%) in chronic moderate asthmatics.

1.3 HYPOTHESES OF THE STUDY

For objectives **1.2.1**, **1.2.2** and **1.2.3**:

The Null Hypothesis (H_0) was set with respect to the short-term effect of an inhaled, short-acting β_2-agonist bronchodilator on chest wall expansion and stated that there would be no significant difference in the chest wall expansion measurements following inhalation of a short acting short-acting β_2-agonist bronchodilator.

The Null Hypothesis (H_0) was set with respect to the short-term effect of an inhaled, short-acting β_2-agonist bronchodilator on the lung function parameters FVC and FEV_1/FVC% and stated that there would be no significant difference in these parameters following inhalation of a short acting short-acting β_2-agonist bronchodilator.

With respect to the short-term effect of the inhaled, short-acting β_2-agonist bronchodilator on FEV_1, the Alternate Hypothesis (H_a) was, however, set based on the report and findings of Haslett *et al*. (2003), Morris and Perkins (2005) and Gina Report (2007). This stated that there would a significant increase in FEV_1 after inhalation of a short-acting β_2-agonist bronchodilator.
The Null Hypothesis (H_0) was set with respect to the short-term effect of SMT and ribcage mobilisation on chest wall expansion and stated that there would be no significant difference in the chest wall expansion measurements following SMT and ribcage mobilisation.

The Null Hypothesis (H_0) was set with respect to the short-term effect of SMT and ribcage mobilisation on the lung function parameters FEV_1, FVC and FEV_1/FVC% and stated that there would be no significant difference in these parameters following SMT and ribcage mobilisation.

The Null Hypothesis (H_0) was set with respect to the short-term effect of a combination of SMT, rib cage mobilisation and an inhaled, short-acting β_2-agonist bronchodilator on chest wall expansion and stated that there would be no significant difference in the chest wall expansion measurements following SMT, ribcage mobilisation and inhalation of a short-acting β_2-agonist bronchodilator.

The Null Hypothesis (H_0) was set with respect to the short-term effect of a combination of SMT, rib cage mobilisation and an inhaled, short-acting β_2-agonist bronchodilator on the lung function parameters FEV_1, FVC and FEV_1/FVC% and stated that there would be no significant difference in these parameters following SMT, ribcage mobilisation and inhalation of a short-acting β_2-agonist bronchodilator.

With respect to the chest wall expansion measurements, the Null Hypothesis (H_0) was set which stated that there would be no significant difference in these measurements between the three groups.

Based on the report and findings of Haslett *et al*. (2003), Morris and Perkins (2005) and Gina Report (2007), the Alternate Hypothesis (H_a) was set which stated that there would a significant increase in FEV_1 after inhalation of a short acting short-acting β_2-agonist bronchodilator as compared to the other two groups.

With respect to the FVC and FEV_1/FVC% parameters, the Null Hypothesis (H_0) was set which stated that there would be no significant difference in these parameters between the three groups.

1.4 SCOPE OF THE STUDY

The results of 45 chronic moderate asthmatics who met all the inclusion criteria of the study are reported in this dissertation. The subjects were divided into three groups of 15 each. Group A received a short-acting β_2-agonist bronchodilator, Group B received SMT of the thoracic spine and ribcage mobilisation and Group C received a combination of SMT, ribcage mobilisation and a short-acting β_2-agonist bronchodilator. Baseline measurements and testing included chest wall expansion

(cm) and lung function parameters (FEV_1, FVC and FEV_1/FVC%). These measurements were repeated 15 minutes post-intervention.

CHAPTER TWO

LITERATURE REVIEW

2.1 INTRODUCTION

Asthma is a serious global health problem as it is a significant burden, not only in terms of health care costs but also due to loss of productivity and reduced participation in family life (Gina Report, 2007). The primary medical management of asthma entails the use of either controller (e.g. corticosteroids) or reliever (e.g. inhaled short-acting β_2-agonist bronchodilators) medication or a combination of both (Rabe and Schmidt, 2001; Gibbon, 2003; Gina Report, 2007). By decreasing muscle tone in both small and large airways in the lungs, bronchodilator medications, therefore, allow for increased airflow and ventilation (Gibbon, 2003; Gina Report, 2007). According to Lane (1991), some patients may desire non-pharmacological therapies to complement their standard pharmacological treatment. Although SMT is one of the more popular complementary therapies in the management of asthma (Nielson *et al*., 1995), its effectiveness is subject to debate since some studies have indicated that SMT may be beneficial as an adjunctive therapy (Gobrin, 1997; Nilsson and Christiansen, 1998; Bronfort *et al.*, 2001; Leboeuf-Yde *et al.*, 2005) while other studies have shown no beneficial effects (Nielsen *et al*., 1995; Jamison *et al*., 1996; Balon *et al*., 1998; Hondras *et al.*, 2000). Furthermore, according to Rosner (2006), none of the studies reviewed excluded the possible masking effects of the medication i.e. the bronchodilator. Kriel (2005) demonstrated an improvement in chest wall expansion and lung function parameters in healthy asymptomatic males following ribcage mobilisation. He recommended that this technique be utilised on symptomatic individuals in order to determine if similar improvement could be achieved.

2.2 RELEVANT ANATOMY OF THE THORACIC SPINE AND RIBCAGE

The kyphotic thoracic spine, with a total composition of 12 vertebrae, commences immediately after the cervical spine and ends at the thoracolumbar area just before L1. According to Panjabi *et al*. (1991) there are three distinct morphological regions in the thoracic spine viz. transitional cervicothoracic and thoracolumbar regions and an intermediate zone which consists of thoracic vertebrae three to nine.

The thoracic cage is formed by part of the vertebral column, 12 pairs of ribs and costal cartilages and the sternum. The ribs and costal cartilages are most significant contributors to the thoracic (rib) cage (Moore and Dalley 2005). The stability of the thoracic spine is substantially enhanced by the ribcage and its articulations (Palastanga *et al*., 2000). The limitations imposed by the structural elements of the ribcage and the vast array of ligamentous and muscular connections further contribute to the stability of this region of the vertebral column (Palastanga *et al*., 2000; Moore and Dalley, 2005). This stability which is essential in the protection of the vital organs, such as the heart and lungs, has further implications for the contribution of thoracic spine stiffness to injuries (Moore and Dalley, 2005).

A typical thoracic vertebra has a body, two transverse costal (rib) facets, two inferior and two superior costal facets, a vertebral foramen, two transverse processes (TVPs), articular processes and a spinous process (SP) (Williams *et al.*, 2005). An atypical thoracic vertebra has a long, almost horizontal SP that is very prominent. It also has tubercles similar to the mamillary and accessory processes of a typical lumbar vertebra (Moore and Dalley, 2005). All thoracic vertebrae are classified as typical vertebrae with the exception of the first thoracic vertebra (T1) and T9-T12 which are known as atypical vertebrae (Moore and Dalley, 2005). The typical body of a thoracic vertebra is heart-shaped and is almost in the form of a waisted cylinder except where the vertebral foramen commences. The anteroposterior (AP) and transverse dimensions of the body are almost equal. Two costal facets are located on either side of the body with one pair at the superior border and one pair at the inferior border. The superior pair lies anterior to the pedicles and is usually larger, while the inferior pair lies anterior to the vertebral notches and is usually smaller. The vertebral foramen of a typical thoracic vertebra is small and circular and the laminae are thick, short, broad and overlap superiorly to inferiorly. The spinous processes (SPs) are long, slender and are angled inferiorly from T5-T8 (Williams *et al.*, 2005). The TVPs, which project from the pediculolaminar junctions in a posterolateral direction, shorten as one moves down the thoracic spine. An anterior oval facet that articulates with the tubercle of the corresponding rib is found on the tip of each of these TVPs (Moore and Dalley, 2005).

The facet joints of the thoracic spine lie principally in the coronal plane allowing for a mainly rotational movement of the thoracic spine (Williams *et al.*, 2005). At T11, the articular processes change in orientation from the thoracic to the lumbar type (the so-called transitional vertebra), but this can also occur at T10 or T12. The superior articular processes (SAPs) in the transitional vertebra face posterolaterally, with the inferior articular processes (IAPs) transversely convex and

directed anterolaterally (Moore and Dalley, 2005). These factors have implications for the biomechanical function (Oxland *et al*., 1992). For example, while the range of motion (ROM) for flexion is less in the thoracic region than in other areas of the spine (White and Panjabi, 1978; Stokes, 2000; Moore and Dalley, 2005), axial rotation is greater than in the lumbar region and almost as great as that of the cervical region (White and Panjabi, 1978).

Ribs are narrow, curved flat bones that form most of the chest wall. They develop by growth in the costochondral cartilages anteriorly and in a posterior growth region close to the vertebral bodies (Stokes, 2000). Appositional bone formation within the ribs appears to continue throughout adult life. Early in life, the transverse plane dimensions of the ribcage become less circular in cross section (Stokes, 2000). Ribs are classified as typical and atypical ribs with ribs three to nine as typical and ribs one, two, ten, 11 and 12 as atypical ribs (Moore and Dalley, 2005). The first rib is broadest and most sharply curved and has a single facet for articulation with the T1 while the second rib is thinner and longer than the first rib and has two facets for articulations with the bodies of the T1 and T2 vertebrae (Moore and Dalley, 2005; Standring, 2005). The tenth and 12^{th} ribs have one facet on their heads while the 11^{th} and 12^{th} ribs are short with no necks and tubercles (Moore and Dalley, 2005; Standring, 2005). Each typical rib has a head, neck, angle and shaft (Palastanga *et al*., 2000; Moore and Dalley, 2005). The head is wedge shaped and has two facets which articulates with the numerically corresponding vertebrae and the vertebrae below it (Moore and Dalley, 2005). The first seven cartilages join the sternum while the eight, ninth and tenth ribs articulate with the cartilages just superior to them (Moore and Dalley, 2005; Standring, 2005).The ribs articulates with the thoracic vertebrae at the superior and inferior demifacets (Moore and Dalley, 2005). The first to seventh ribs articulate through their costal cartilages with the lateral borders of the sternum (Moore and Dalley, 2005). Ribs with their costal cartilages are separated by intercostal spaces that are occupied by the intercostal muscles, vessels and nerves (Moore and Agur, 1996).

The intercostal spaces contain three layers of intercostal muscles. The superficial layer is the external intercostal muscle, the middle layer is the internal intercostal muscle and the deepest layer is the innermost intercostal muscle. The external and innermost intercostals act to elevate the rib cage while the internal intercostals depress the ribs. Costotransverse joints are synovial joints (Palastanga *et al*., 2000; Moore and Dalley, 2005; Standring 2005). The tubercle of a typical rib articulates with the transverse costal facet of its own vertebrae (Palastanga *et al*., 2000; Moore and Dalley, 2005; Standring, 2005). The 11^{th} and 12^{th} ribs lack this articulation (Standring, 2005). In the

upper five or six joints, the articular surfaces are reciprocally curved, but below this they are flatter (Standring, 2005). The joints are surrounded by capsular, superior and lateral costotransverse and accessory ligaments (Moore and Dalley, 2005; Standring 2005).These joints have a thin fibrous capsule which has a synovial lining (Standring, 2005).

2.3 PULMONARY RESPIRATION AND VENTILATION

Respiration in man may be defined as the process concerned with gaseous exchange between Man and his environment, starting with the inhalation of oxygen and ending with the exhalation of carbon dioxide (Moxham and Costello, 1994). Respiration is unique, in that, of all the vital functions, it alone is regulated not only by autonomic centres located in the brainstem but also by voluntary signals initiated in the cortex (American Thoracic Society, 1999). The process of respiration involves two components: 1) pulmonary or external respiration which is the absorption of oxygen and the removal of carbon dioxide from the body as a whole (Ganong, 1995; Guyton and Hall, 2000; Palastanga *et al*., 2000) and 2) internal respiration which results in the utilization of oxygen and the production of carbon dioxide by cells (Ganong, 1995; Guyton and Hall, 2000). The respiratory system, which ensures the functioning of the above process, is comprised of the lungs, a pump that ventilates the lungs, the central nervous system and the pulmonary circulation (Ganong, 1995; Moore and Dalley, 2005). The pump consists of the chest wall and its associated respiratory muscles which increase and decrease the size of the thoracic cavity (Ganong, 1995; Guyton and Hall, 2000). At rest, a normal human breathes 12-15 times a minute (Ganong, 1995; The Merck Manual, 2003) with six to eight litres of air per minute being inspired and expired (Ganong, 1995; The Merck Manual, 2003). The total lung surface area available for respiratory exchange is about 70 m^2 (Palastanga *et al*., 2000).

During respiration the volume of the thoracic cage changes due to the movement of the diaphragm, ribs and sternum (Guyton and Hall, 2000; Palastanga *et al*., 2000). The ribs and their costal cartilages act as levers which move up and down (Palastanga *et al*., 2000; Stokes, 2000). This movement is dependent on several factors including the rib length and whether the rib articulates directly with the sternum or not (Palastanga *et al*., 2000). The movement of the upper ribs (i.e. the second to the fifth ribs) around the axis along their necks results in the raising of their anterior ends and the body of the sternum. The body of the sternum is therefore lifted upwards and outwards causing an increase in the AP diameter of the thorax (Palastanga, 2000). This movement is referred to as the pump handle movement (Palastanga *et al*., 2000; Stokes, 2000; Gatterman, 2003; Moore and Dalley, 2005). The movement of the eighth to the tenth ribs results in an outward

and upward movement of their anterior ends (Palastanga *et al*., 2000). The lateral excursion of the ribs and their costal cartilages causes a widening of the infrasternal angle and a consequent increase in the transverse diameter of the thorax (Palastanga *et al*., 2000; Stokes, 2000; Gatterman, 2003). The upward and outward movement of the shaft of the rib is referred to as the bucket handle movement (Palastanga *et al*., 2000). The intermediate ribs six and seven show both pump handle and bucket handle types of movement (Palastanga *et al*., 2000). Since the 11^{th} and 12^{th} ribs are not attached anteriorly they, therefore, have little influence on increasing the transverse diameter of the thorax (Palastanga *et al*., 2000). During expiration the reverse movements are true with a decrease in both the AP and transverse diameter of the thorax (Guyton and Hall, 2000; Palastanga *et al*., 2000).

According to Gatterman (2003), the mobility of the ribs varies considerably in that the first few ribs are more fixed than the others due to the weight of the upper extremities and the strain of the ribs beneath while increased mobility is seen in the intermediate region of the thoracic cage down to the ninth and tenth ribs. This is supported by Moore and Agur (1996) who state that the first seven ribs are joined to the sternum, the next three have their costal cartilages joined to the rib above them, while the eleventh and twelfth ribs are ‘floating’ or ‘free’ ribs as their costal cartilages end in the abdominal muscle wall. By virtue of its attachment to the thoracic cage, the motion of the spine may influence the motion of the thoracic cage (Stokes, 2000).

The term ventilation refers to the mechanical process of moving air into and out of the lungs (McArdle *et al.*, 1996), a process which is achieved by the diaphragm in association with the respiratory muscles which act as a pump on the chest wall, and is under the control of the central nervous system (Stokes, 2000; Ganong, 1995). Davies *et al.* (2001) state that the respiratory muscles, the chest wall and the diaphragm act on the lungs to cause inspiration as well as expiration. They also credit the respiratory muscles with acting to inflate and deflate the lungs through the generation of a pressure difference, which causes movement of air through the airways. Therefore, movement of the respiratory muscles and the chest wall as a whole, results in a corresponding movement within the lungs. Gonzales *et al*. (1999) had previously reported that restriction in chest wall expansion would result in decrements in pulmonary function. Hypertonicity of the intercostals muscles and alteration in rib biomechanics are, therefore, likely to have an effect on the optimal functioning of the lungs.

Gatterman (2003) and Standring (2005) state that the respiratory muscles may be divided into two groups according to the role they play in respiration viz. the inspiratory and expiratory muscles, with a further division into primary and accessory muscles. The primary muscles of inspiration include the diaphragm and external intercostal muscles. The accessory muscles include the sternocleidomastoid, scalenius anterior, scalenius medius, scalenius posterior, serratus anterior, serratus posterior, latissimus dorsi, pectoralis major, pectoralis minor and the superior fibres of the iliocostalis muscles. The primary muscles of expiration include the internal intercostal muscles and the diaphragm, while the accessory muscles include the external abdominal oblique, internal abdominal oblique, transverse abdominal, latissimus dorsi, serratus posterior inferior and the quadratus lumborum muscles. One of the primary roles of the intercostal muscles is to stiffen the chest wall, preventing paradoxical motion during descent of the diaphragm in inspiration (Palastanga *et al*., 2000; Standring, 2005). The external intercostal muscles attach at an oblique angle from rib to rib (Ganong, 1995; Palastanga *et al*., 2000). The upper ribs pivot as if hinged at the back so that when the external intercostal muscles contract they elevate the lower ribs with the sternum being pushed outward resulting in an increase in the AP diameter of the chest (Ganong, 1995). Forced expiration and a decrease in the intrathoracic volume occur when the expiratory muscles contract (Ganong, 1995). Since the internal intercostals muscles pass obliquely downward and posteriorly from rib to rib they, therefore, pull the ribcage downward when they contract (Ganong, 1995). They are aided by the contractions of the anterior abdominal wall muscles which also pull the ribcage downward and inward and by the increase in the intra-abdominal pressure which pushes the diaphragm upwards (Ganong, 1995).

Powers and Howley (1997) state that during quiet, normal, breathing inspiration is controlled primarily by the diaphragm, but during exercise or increased physical activity, accessory muscles are recruited. During quiet inspiration, the movement of the diaphragm accounts for 75% of the change in the intrathoracic volume (Ganong, 1995). The costal portion of the diaphragm which is attached to the ribs is responsible for ventilation (Standring, 2005). During exhalation, the lateral portions of the diaphragm are in contact with the sides of the ribcage and become separated from the ribs during inhalation, resulting in a piston-like movement that expands the lungs (Standring, 2005).

The respiratory centre in the medulla oblongata, consisting of dorsal and ventral groups of neurons, controls spontaneous respiration. Efferent fibres from the respiratory centre pass in the ventral and lateral parts of the spinal cord to the motor neurons that control the respiratory muscles, while the

vagus nerve supplies the accessory muscles involved in respiration. Descending pathways to the inspiratory muscles inhibit the expiratory muscles via inhibitory interneurons in the brain stem thereby preventing the muscles from contracting and conflicting with the inspiratory muscles. The converse occurs when the expiratory muscles become active. Also, an indication that higher centres of the brain have an influence on the medullary centres controlling respiration can be demonstrated through the fact that emotional factors as well as pain have an influence on inspiration (Rang *et al.*, 2007).

McArdle *et al*. (1996) state that any change in the volume of the thoracic cavity results in a corresponding change in lung volume. The lungs depend on accessory means for altering their volume because they contain no muscles and this volume is altered during inspiration and expiration by the action of voluntary muscles. According to Moore and Dalley (2005) and Standring (2005), each lung is surrounded and enclosed by pleurae, the inner layer called the visceral pleura which covers the surface of the lung (except the hilar region) and the parietal pleura which is adherent to the chest wall, mediastinum and diaphragm.

Inspiration is an active process that results in the contraction of the inspiratory muscles which increases the intrathoracic volume (Ganong, 1995). At the commencement of inspiration, the intrapleural pressure at the base of the lungs is normally about -2.5 mmHg which decreases to about -6 mmHg as inspiration ends (Ganong, 1995). This allows for the lungs to be pulled into a more expanded position (Ganong, 1995). The pressure in the airways becomes slightly negative allowing for air to flow into the lungs. At the end of inspiration the lung recoil begins to pull the chest back to the expiratory position where the recoil pressures of the lungs and chest wall balance (Ganong, 1995). The pressure in the airways becomes slightly positive resulting in the flow of air out of the lungs (Ganong, 1995). The increased loads on the inspiratory muscles in severe asthma has been recognised for many years, but it remains uncertain whether this leads to increased force and endurance in chronic asthma or to muscle fatigue in severe acute attacks (Pride, 1992). Gross inspiratory weakness was observed in a few obese women with asthma who had been on chronic high doses of oral corticosteroids (Pride, 1992).

Expiration is predominantly a passive process during rest and light exercise since there is no muscle contraction which causes a decrease in the intrathoracic volume (Ganong, 1995). There is, however, some inspiratory muscle contraction in the early phase of expiration resulting in a slowing down action on the recoil forces which then slows expiration (Ganong, 1995). During ventilation in

heavy exercise, the internal intercostal and abdominal muscles act powerfully on the ribs and abdominal cavity to facilitate the reduction of the thoracic dimensions. This suggests that the muscles of the ribs are capable of more rapid action than the diaphragm and the abdominal muscles (McArdle *et al*., 1996)

2.4 LUNG FUNCTION PARAMETERS

According to Johns and Pierce (2007), much can be learned about the mechanical properties of the lungs from measurements of forced expiration and inspiration. FVC is the volume of air expired with maximal force (Ganong, 1995). It is usually measured along with expiratory flow rates in simple spirometry (Ganong, 1995). During the FVC maneuver, terminal airways can close prematurely, trapping gas distally and preventing its measurement by the spirometer (Ganong, 1995; The Merck Manual, 2003). Patients with obstructive lung disease (e.g. emphysema) (Haslett *et al.,* 2003) usually have a normal or only slightly decreased FVC while those with restrictive lung disease (e.g. asbestosis) (The Merck Manual, 2003) have a decreased FVC (Ganong, 1995; The Merck Manual, 2003).

Forced expiratory volume in one second (FEV_1) is the volume of air forcefully expired during the first second after a full breath (Johns and Pierce, 2007) and normally accounts for greater than 75% of the FVC (The Merck Manual, 2003; Ganong, 1995). This value is recorded both as an absolute value and as a percentage of the FVC (FEV_1/FVC %) (Ganong, 1995; The Merck Manual, 2003; Johns and Pierce, 2007) and is regarded as “the most reproducible value derived from spirometry” (Jamison *et al*., 1986). The FEV_1 is reduced in obstructive lung disease because of increased airway resistance (Ganong, 1995; The Merck Manual, 2003) while in restrictive lung disease it is because of the low vital capacity (VC) (Ganong, 1995).

FEV_1/FVC% provides a clinically useful index of airflow limitation (Ganong, 1995; Johns and Pierce, 2007). In healthy patients the FEV_1/FVC% is usually around 70% (Johns and Pierce, 2007). In obstructive lung disease FEV_1/FVC% decreases and can be as low as 20-30% in severe cases (Haslett *et al.,* 2003). Restrictive disorders, on the other hand, have a near normal FEV_1/FVC% (Ganong, 1995; Johns and Pierce, 2007).

Other lung function parameters include VC which is the volume of gas that can be expelled from the lungs from a position of full inspiration, with no limit to the duration of expiration (Guyton and Hall, 2000). The normal VC is usually about 4600 ml (Guyton and Hall, 2000). Tidal volume is the volume of air inspired or expired with each normal breath (Guyton and Hall, 2000) and is about 500 ml in the average young adult male (Guyton and Hall, 2000). Residual volume (RV) is the volume of air remaining in the lungs after the most forceful expiration (Ganong, 1995; Guyton and Hall, 2000) and averages about 1200 ml (Guyton and Hall, 2000).

2.5 MECHANORECEPTORS

The primary mechanoreceptors in the chest are the muscle spindle endings and tendon organs of the respiratory muscles and the joint proprioceptors (Powers and Howley, 1997). Muscle spindles are primarily influenced by changes in length and are responsible for reflex contraction of the skeletal muscles in response to stretching (Powers and Howley, 1997). Afferent information from these receptors in the chest wall is carried in the anterior columns of the spinal reticular pathway and terminates in the region of the respiratory centres in the medulla. Muscle receptor afferents play a role in the level and timing of respiratory activity (Duron, 1981). Muscle spindles are responsible for the reflex contraction of skeletal muscles that occurs during rapid stretching (Powers and Howley, 1997). The function of the muscle spindle is to assist in the regulation of movement and to maintain posture. This is accomplished by its spindle's ability to respond to changes in length of skeletal muscle fibres. The Golgi tendon organs (GTOs) continuously monitor tension produced by muscle contraction (Powers and Howley, 1997). Earlier, Duron (1981) stated that GTOs sense changes in the force of contraction exerted by the muscles of respiration and are involved in monitoring the force of muscle contraction during breathing at rest or with a respiratory load.

The respiratory muscles, of which the intercostal muscles form an integral part, are innervated by a variety of sensory receptors including muscle spindles. Afferent activity from them involves the spinal and supraspinal reflexes (Bolsher *et al.,* 1988). According to Powers and Howley (1997), in order for the nervous system to properly control skeletal muscle movements, it must receive continuous sensory feedback from the contractile muscles. This sensory feedback includes (1) information concerning the tension developed by a muscle and (2) an account of muscle length (Powers and Howley, 1997). Feedback of afferent information from the lung and chest wall mechanoreceptors provides respiratory motor and pre-motor neurons with important information regarding the status of the ventilatory pump as well as changes in length and force of contraction of

the respiratory muscles (Guyton and Hall, 2000). These signals allow adjustments to be made in the level and pattern of brainstem respiratory motor activity to compensate for changes in respiratory muscle function (Ganong, 1995; Guyton and Hall, 2000). Projections to the brain of afferent signals from mechanoreceptors in the joints, tendons and muscles of the chest all appear to play a role in shaping respiratory sensations (Gandevia and Macefield, 1989). Specifically afferents from the intercostal muscles have been shown to project to the cerebral cortex and contribute to proprioception and kinaesthesia (Homma *et al.,* 1988). Joint proprioceptors sense the degree of chest wall movement and may also influence the level and timing of respiratory activity (Duron, 1981). These mechanoreceptors may also be important in the sensation of dyspnoea (Manning and Schwartzstein, 1995).

In their in vivo studies of cat intercostal muscles, Holt *et al.* (2002) showed that there are three populations of intercostal muscle mechanoreceptors: primary muscle spindles, secondary muscle spindles and GTOs. It was found that both primary and secondary degree muscle spindles were evenly distributed within the intercostal muscle space, while the GTOs were localized along the rib borders. It is quite possible that similar mechanoreceptors and their distribution may be found in the intercostal muscles of humans although there are no human studies that show this.

2.6 MOBILISATION

Triano (2005) defined joint mobilisation as “cyclic application of joint loads at lower velocity” which is administered by a clinician for the purposes of increasing overall range of motion (ROM). A later definition by The Canadian Orthopractic Manual Therapy Association (2002) states that mobilisation is a gentle, repetitive, passive movement of graded amplitude aimed at restoring mobility and function and reducing pain in a joint and surrounding tissue. Maitland *et al*. (2001) are of the view that mobilisation of a joint is a passive movement performed in such a manner (particularly in relation to speed of movement) that it is, at all times within the ability of the patient to prevent the movement if he or she chooses. The indications for joint mobilisation include pain, subluxation or dislocation and joint hypomobility (Flynn, 1996).

2.6.1 Spinal Mobilisation

There are few studies which have investigated the role of joint mobilisation on pain reduction and ROM. Cassidy *et al.* (1992) conducted a randomised, controlled clinical trial in order to compare the immediate results of spinal manipulation (n = 52) versus spinal mobilisation (n = 48) on pain and ROM of the cervical spine. The authors concluded that both treatments increased cervical spinal

ROM to a similar degree. A similar finding was reported by Pillay (2001) who investigated the effectiveness of spinal manipulation (*n* = 30) compared to passive oscillatory mobilisation (*n* = 30) in the management of chronic mechanical thoracic spine pain. The author concluded that both spinal manipulation and passive oscillatory mobilisation are effective interventions in the treatment of chronic mechanical thoracic spine pain. Dimopoulos (2002) conducted a study similar to that of Pillay (2001) in 40 subjects with chronic thoracic spine dysfunction divided equally into two groups. Subjects in Group 1 received thoracic SMT while those in Group 2 received passive thoracic oscillatory mobilisation. He concluded that both SMT and passive thoracic oscillatory mobilisation were effective manual interventions for the reduction in the patients pain response, pain intensity and ROM measurements.

2.6.2 Rib Mobilisation

Studies on extra-spinal mobilisation are scant but the results reported appear to be favourable. Hightower *et al.* (1999) evaluated the effects of ribcage mobilisation and respiratory muscle stretching compared to a control group on VC and chest wall expansion in 14 adults aged 58 to 83 years. The results indicated that there was an improvement in xiphoid and axillary chest wall expansion measurements (cm) following manual stretching and rib mobilisation compared to a control group. It is, however, difficult to ascertain the contribution of the rib mobilisation alone to this result. A study by Kriel (2005) on ribcage mobilisation in healthy asymptomatic males revealed that $FEV_{1,}$ FVC and FEV_1/FVC% improved post-mobilisation compared to a placebo (sham laser) and a control group (no intervention). He, therefore, recommended that future studies investigate the effect of ribcage mobilisation on individuals with symptomatic lung disease e.g. asthma. Kriel (2005) attributed his findings to an increase movement of the ribs following mobilisation and a possible response by the intercostal mechanoreceptors. These responses may have resulted due to adjustments to chest wall movement or a reflex contraction as alluded to by Powers and Howley (1997). Earlier, Duron (1981) stated that the effects of movement on the chest wall and its components (the ribs, respiratory muscles and joints) by the application of rib mobilisations, may lead to a response by the mechanoreceptors.

2.7 SPINAL MANIPULATION

Manipulation of the spine has been practiced since ancient times (Triano, 2001). Hippocrates, the father of medicine, had also practiced spinal manipulation as a means of treating back pain (Wiese and Callender, 2005). SMT gained popularity in Europe during the 18th century and was practiced predominantly by manual therapists called bonesetters (Wilson and Keating Jr, 2007). In 1895, the

first chiropractic manipulation or adjustment was performed by D.D. Palmer (Wilson and Keating Jr, 2007).

2.7.1 Spinal Manipulation and Thoracic Spine Mobility

Gatterman (2003) describes SMT as the use of a short lever, high-velocity thrust of controlled amplitude on spinal articulations with the aim of restoring mobility to the individual articulations. This results in improved flexibility and increased joint mobility (Herzog *et al*., 1988; Gal *et al*., 1994; Herzog, 2000; Gatterman *et al*., 2001; Gatterman, 2003). A more recent definition by Vernon and Mrozek (2005) described manipulation or adjustment as moving of the joints of the spine beyond a person's clinical physiological ROM, using a fast low amplitude thrust often resulting in an audible click or pop, the so-called joint cavitation (Shekelle, 1994; Vernon and Mrozek, 2005). The actual manipulation is not performed at the limit of the clinical physiological range; rather it is performed at a point slightly beyond this range called the closed packed position. Schiller (2001) and Dimopoulos (2002) demonstrated that thoracic ROM increased following SMT in individuals with mechanical thoracic spine pain. An increase in thoracic spine ROM was also reported by Sood (2008) in his study to determine the effect of SMT on bowling speed in healthy, asymptomatic Action Cricket fast bowlers.

2.7.2 Neurophysiological, Inflammatory and Somatovisceral Responses to Spinal Manipulation

Nielsen *et al*. (1995) reported that SMT may have some physiological effect on the autonomic nervous and neuroendocrine systems. A reduction in blood pressure and changes in adrenal and renal activity was observed in rats following mechanical stimulation of the spinal afferent nerves (Sato and Swenson, 1984). In another animal study (rabbits), DeBoer *et al*. (1988) demonstrated inhibition of the gastrointestinal myoelectric activity following thoracic spine manipulation. Kaliner *et al*. (1982) stated that an imbalance in the autonomic nervous system could contribute to airway dysfunction in asthmatic patients. Degranulation of mast cells and basophils may occur in response to autonomic dysfunction since these cells contain receptors belonging to the autonomic nervous system (Garrity *et al*., 1985). Garrity *et al*. (1985) had also demonstrated that sympathetic stimulation may decrease degranulation of mast cells under controlled conditions. Enhanced oxidative burst activity of neutrophils and monocytes from individuals who received SMT was observed by Brennan *et al*. (1991). The plasma concentrations of substance P, a neuropeptide which is a known enhancer of the inflammatory process in asthma, was reduced following SMT. An

in vitro study has shown that, at low concentrations, substance P is able to prime phagocytes for augmented oxidative burst activity (Brennan *et al*., 1992). Hayek (2002) reported an increase in IgA concentration and decrease in cortisol concentration in asthmatic patients who received SMT. The increase in IgA concentration would be immuno-protective as the risk of pathogen invasion through the airways would be reduced. The function of the immune system would be further enhanced in these patients through the reduction of the immuno-suppressant cortisol.

In humans, somatovisceral effects have been reported post-SMT (Nielsen *et al*., 1995). An elevation in the concentration of β-endorphins was found in individuals who received SMT (Vernon *et al*., 1986). Sensory input from paraspinal tissues can evoke visceral reflexes affecting the sympathetic nervous system and may alter end-organ function (Pickar, 2002). In 1963, Homewood suggested that restrictions in the upper thoracic segments may result in inhibition of normal sympathetic impulses through the pulmonary plexus, resulting in vasodilation, bronchial constriction and over activity of mucous glands, being the symptoms in catarrhal complexes and asthma.

2.8 ASTHMA

2.8.1 Description and Epidemiology of Asthma

Asthma is derived from the Greek word ‘Aazein’ which means sharp breath (The Asthma and Allergy Foundation of America, 2005; Asthma History, 2008). It is a chronic inflammatory condition of the airways that is usually allergic in origin (Gibbon, 2003). Since asthma cannot be cured but can be controlled, this requires many patients to take preventative medication on a daily basis (National Guideline for the Management of Asthma in Adults at Primary Level, 2002).

Asthma is one of the most common chronic diseases worldwide including the United States and other industrialized countries such as Canada, England, Australia, Germany and New Zealand (Marik *et al.,* 2002; Camargo, 2006; Gina Report, 2007). Some 300 million people currently suffer from asthma (World Health Organisation, 2008). Not only is asthma the most common chronic disease among children (World Health Organisation 2008), it is also the most common cause of hospitalization for children in the United States (Morris and Perkins, 2005). The prevalence of asthma in industrialized countries varies from 0.7% to 6.3% (Camargo, 2006), but that of severe asthma is higher with ranges from 2% to 10% (Morris and Perkins, 2005).

Asthma symptoms can begin at any age although the usual period of commencement is early childhood (e.g. 80-90% by age six years) (Camargo, 2006). Children younger than ten years

constitute approximately 50% of all cases (Camargo, 2006) where the male-to-female ratio is 2:1. Between the ages of 18 and 54 years this ratio is reversed, with women being affected twice as often as men (Camargo, 2006). Mortality statistics from the United States indicate that in 1998 a total of 5438 deaths were due to asthma (Canaday and Collins, 2004). The self-reported prevalence of asthma in South Africa is 7% in males and 9% in females (National Guideline for the Management of Asthma in Adults at Primary Level, 2002) which is higher than the international figures provided by Camargo (2006), but in keeping with the more recent statistics reported by (Morris and Perkins, 2005). The accuracy of statistics about asthma is, however, limited by confounding factors in patients older than age 35 years, changes in the International Classification of Diseases, the lower confirmation rates at autopsy, and problems related to the interpretation of death certificates (Busse and Lemanske, 2001).

The World Health Organisation has estimated that 15 million disability adjusted life years are lost annually due to asthma, representing one percent of the total global disease burden (Masoli *et al.,* 2004). The monetary costs of asthma, as estimated in a variety of health care systems including those of the United States (Weiss *et al.,* 1992) and the United Kingdom (Action Asthma, 1990) are substantial.

2.8.2 Aetiology and Synopsis of the Pathophysiology of Asthma

Asthma is a syndrome that is characterized by clinical features that centre on disorders of lung function; the diagnosis of asthma is, therefore, best made by results obtained from lung function tests (Eidelman and Irvin, 2000; Kaminsky and Irvin, 2002; National Asthma Education, 2002). The development of asthma is multifactorial and may depend on interactions between multiple susceptibility genes and environmental factors (Maddox and Schwartz, 2002; Gina Report, 2007). Furthermore, asthma is a chronic inflammatory disorder of the airways in which many cells and cellular elements are involved viz. mast cells, eosinophils, T-lymphocytes, macrophages, neutrophils and epithelial cells (Rabe and Schmidt, 2001; Marik *et al*., 2002; Bukstein *et al*., 2006. In susceptible individuals, this inflammation causes recurrent episodes of wheezing, breathlessness, chest tightness and coughing, particularly at night or in the early morning (Luskin, 2005). These episodes are usually associated with widespread but variable airflow obstruction that is often reversible either spontaneously or with treatment. The inflammation also causes an associated increase in the existing bronchial responsiveness to a variety of stimuli (Luskin, 2005; Camargo, 2006).

Clark *et al*. (1992), Miller (2001) and Nauta *et al.* (2008) suggested that increased resistance to airflow may be related to environmental factors, especially inhaled pollutants in concentrations that do not affect the majority of persons. Furthermore, physical and chemical stimuli, inhaled antigens, cigarette smoke (Nauta *et al.,* 2008), emotional distress (Luskin, 2005), drugs and exercise (National Asthma education, 2002; Gina Report, 2007) may lead to hyperresponsiveness of the airways and specific antigen-antibody reactions (National Asthma Education, 2002; Gina Report, 2007).

The pathophysiology of asthma is complex and basically involves the following components: (1) airway inflammation (2) intermittent airflow obstruction and (3) bronchial hyperresponsiveness (Rabe and Schmidt, 2001; Corrigan, 2008). The mechanism of inflammation in asthma may be acute, subacute or chronic, and the presence of airway edema and mucus secretion also contributes to airflow obstruction and bronchial reactivity (Camargo, 2006). The degree of airway hyperresponsiveness generally correlates with the clinical severity of asthma (Corrigan, 2008).

Findings of abnormal histopathologic lesions including swelling, sloughing of the epithelial cells and inflammatory cell infiltration may occur at any stage of asthma as these have been reported not only in autopsy studies of severe asthma cases, but also in patients with very mild asthma who have underwent research bronchoscopy (Camargo, 2006). Airway wall remodelling may occur due to including goblet cell hyperplasia, subepithelial fibrosis, smooth muscle cell and myofibroblast hyperplasia (Corrigan, 2008). Interactions between the neural mechanisms, inflammatory cells, chemical mediators such as interleukins, leukotrienes, and prostaglandins, and intrinsic abnormalities of the arachidonic acid pathway and smooth muscle cells all contribute to the multifactorial nature of asthma (Martinez, 2007; Corrigan, 2008).

Acute bronchoconstriction, airway edema, chronic mucous plug formation and airway remodeling are primarily responsible for acute airway obstruction (Barnes, 1998). Acute bronchoconstriction is the consequence of immunoglobulin E–dependent mediator release upon exposure to aeroallergens and is the primary component of the early asthmatic response (Miller, 2001; Corrigan, 2008). Airway edema occurs 6-24 hours following exposure to an allergen and is referred to as the late asthmatic response (Miller, 2001). Chronic mucous plug formation consists of an exudate of serum proteins and cell debris that may take weeks to resolve (Miller, 2001). Airway remodeling is associated with structural changes as a result of prolonged inflammation and may profoundly affect the extent of reversibility of airway obstruction (Kumar *et al.,* 2003; Corrigan, 2008). With repeated episodes of clinically important airway narrowing, generalized thickening of

the airways occurs as a result of smooth muscle hyperplasia, postinflammatory thickening of the bronchial basement membranes and mucous gland hypertrophy (Miller, 2001; Kumar *et al.,* 2003; Corrigan, 2008).

2.8.3 Clinical Features of Asthma

The symptoms of asthma may manifest suddenly or gradually (National Guideline for the Management of Asthma in Adult Asthma at Primary Level, 2002; Gina report, 2007). Sufferers typically complain of a tight chest, wheeze and sputum production (National Guideline for the Management of Asthma in Adult Asthma at Primary Level, 2002). The symptoms of each asthmatic sufferer differ greatly in frequency and degree. The wheeze is particular noticeable during expiration (The Merck Manual, 2003). Some asthmatics are symptom-free most of the time, with an occasional episode that is mild and brief (Gobrin, 1997; Gina report, 2007). The general clinical features of asthma are recorded in **Table 2.1**. Several criteria have to be fulfilled to enable one to make a diagnosis of chronic asthma as shown in **Table 2.2**.

Table 2.1 The general clinical features of asthma*

Symptoms	Signs
Breathlessness	Barrel chest
Cough especially at night	Indrawn costal margins
Chest tightness	High pitched wheeze
Thick sputum production	Pulsus paradoxus
Anxiety	Use of accessory respiratory muscles
	Ala flaring of the nostrils

*** from Haslett *et al*. (2003); Canaday and Collins (2004)**

Table 2.2 The diagnosis of chronic asthma*

Chronic mild asthma	Chronic moderate asthma	Chronic severe asthma
Cough	Cough	Cough
Tight chest	Tight chest	Tight chest
Wheeze for 2-4 weeks	Wheeze for > 4 weeks	Continuous wheeze
Night symptoms for 2-4 months	Night symptoms for > 4 months	Frequent night symptoms
PEF > 80%.	PEF 60-80%	PEF < 60 %

*** from National Guideline for the Management of Asthma in Adults at Primary Level (2002), Gina Report (2007)**
PEF = Peak expiratory flow

There are multiple factors implicated in the triggering of an asthma attack. The common causes are cigarette smoke (Miller, 2001; Gina Report, 2007; World Health Organisation, 2008), air pollutants (Miller, 2001; Gina Report, 2007; World Health Organisation, 2008), allergens (Miller, 2001; Gina Report, 2007), dust from pet dander (Miller, 2001; Gina Report, 2007), cold air (Miller, 2001; Gina

Report, 2007), emotion air (Miller, 2001; Gina Report, 2007; World Health Organisation, 2008) and exercise (Kumar *et al.,* 2003). Drugs such as non-steroidal anti-inflammatory drugs (NSAIDs) and the role of genetic factors and the development of the immune system are being scrutinized (Kumar *et al.,* 2003).

2.8.4 Investigations in Asthma

2.8.4.1 Pulmonary Function Testing (Spirometry)

Spirometry is the recommended method of measuring airflow limitation and reversibility in order to establish a diagnosis of asthma (Haslett *et al.,* 2003; Morris and Perkins, 2005; Camargo, 2006; Gina Report, 2007). The American Association for Respiratory Care (2008) includes the need to assess the change in lung function following the administration of a therapy as one of the indications for the use of spirometry which primarily measures the FVC and the FEV_1 (Gina Report, 2007). Recently spirometry, a simple and useful diagnostic tool, has become increasingly available (Clark *et al*., 1992). It also enables the patient to understand the periodicity and provoking factors of their asthma and the therapeutic response to their disease (Prior and Cochrane, 1980). Although spirometry is reproducible, it is effort-dependent (Gina Report, 2007). Therefore, proper instructions on how to use the spirometer and performance of the test must be given to the patients (Gina Report, 2007). The Gina Report (2007) recommends that the highest value of three readings should be recorded.

The hallmark of airway obstruction is a reduction in FEV_1 (Jamison *et al*., 1986) and the ratio of FEV_1 to the FVC (Canaday and Collins, 2004). A reduced ratio of FEV_1 to FVC, when compared with predicted values, demonstrates the presence of airway obstruction (Gina Report, 2007). In asthma the FEV_1 is usually decreased, the FVC is usually normal and the ratio FEV_1/FVC % is decreased (Gina Report, 2007; National Asthma Council of Australia, 2008). The prolongation of expiratory flow rates is increased by bronchospasm (in asthma). Berkow (1992), Haslett *et al.* (2003), Morris and Perkins (2005) and the Gina Report (2007) all state that spirometry measurements should be performed before and after inhalation of a short-acting bronchodilator in all patients in whom the diagnosis of asthma is considered. Reversibility is demonstrated by an increase of 12% or 200 ml after the administration of a short-acting inhaled bronchodilator (Haslett *et al.,* 2003; Morris and Perkins, 2005; Gina Report, 2007). In patients with airway obstruction, the absence of a response to a single exposure to a bronchodilator does not preclude a beneficial response to maintenance therapy (The Merck Manual, 2003). FEV_1 as a measure of asthma

severity has advantages compared to peak expiratory flow (PEF). These include greater accuracy, less effort dependence, better reproducibility, and for some spirometers the availability of real-time graphics and quality assurance checks to confirm reliability of the results (Gardner *et al.,* 1992; Enright *et al.,* 1994).

The diagnosis of asthma, however, cannot be based on spirometry findings alone because many other diseases are associated with obstructive spirometry indices (Morris and Perkins, 2005; Gina Report, 2007).

2.8.4.2 Peak Expiratory Flow Monitoring

PEF monitoring is designed for ongoing monitoring of asthmatic patients. Peak flow meters are relatively inexpensive, portable and ideal for patients to use in a home setting (Gina Report, 2007). The test is simple to perform and the results are a quantitative and reproducible measure of airflow obstruction (Berkow, 1992; Morris and Perkins, 2005; Gina Report, 2007). It can be used for short-term monitoring, exacerbation management and daily long-term monitoring (Morris and Perkins, 2005; Gina Report, 2007). A 60 $L.min^{-1}$ (or 20% or more of pre-bronchodilator PEF) improvement after inhalation of a bronchodilator (Dekker *et al*., 1992) or diurnal variation in PEF of more than 20% (with twice daily readings, more than 10% (Boezen *et al*., 1994)) suggests a diagnosis of asthma. A PEF less than 50% of predicted (or of personal best) represents severe obstruction. When a patient's best PEF is 200 $L.min^{-1}$, a drop to 120 $L.min^{-1}$ (i.e. 60% of the personal best) may signify severe, potentially life threatening obstruction (Emond *et al.,* 1997).

2.8.4.3 Arterial Blood Gas Pressures

The measurements of arterial blood gas (ABG) pressures are indispensable in the management of patients with acute severe asthma (Haslett *et al.,* 2003; Camargo, 2006). This test may reveal dangerous levels of hypoxemia or hypercarbia secondary to hypoventilation; typically, results are consistent with respiratory alkalosis (Camargo, 2006). Because of the accuracy and utility of pulse oximetry, only patients whose oxygenation is not restored to over 90% with oxygen therapy require an ABG (Morris and Perkins, 2005).

2.8.4.4 Laboratory Studies

Laboratory studies are not routinely indicated for asthma but may be used to exclude other diagnoses (Morris and Perkins, 2005). Although, blood eosinophilia greater than 4% or 300-

400.mL^{-1} supports the diagnosis of asthma (Morris and Perkins, 2005), an absence of this finding is not exclusionary (Berkow, 1992; Morris and Perkins, 2005). Total serum immunoglobulin E (IgE) concentrations greater than 100 IU are frequently observed in patients experiencing allergic reactions, but this finding is not specific for asthma and may be observed in patients with other conditions (e.g., allergic bronchopulmonary aspergillosis, Churg-Strauss syndrome) (Morris and Perkins, 2005). A normal total serum IgE concentration does not exclude the diagnosis of asthma (Morris and Perkins, 2005). The British Thoracic Society recommends the use of sputum eosinophilia determinations to guide asthma therapy and to monitor control (Morris and Perkins, 2005). An improvement in asthma control, a decrease in hospitalizations and a decrease in exacerbation of symptoms were noted in patients in whom sputum-guided therapy was used (Morris and Perkins, 2005).

Exhaled nitric oxide (NO) analysis has been utilized to predict airway inflammation and asthmatic control. It is, however, technically more complex and not routinely used in the monitoring of patients with asthma (Morris and Perkins, 2005). Serum theophylline levels are helpful in monitoring patient compliance and excluding inadvertent theophylline toxicity (Camargo, 2006).

2.8.4.5 Chest Radiography

Chest radiography may be helpful in suggesting a diagnosis of asthma despite normal findings or features of hyperinflation seen in the x-rays of most patients (Berkow, 1992; Morris and Perkins, 2005; Camargo, 2006). Furthermore, chest radiography of patients with acute asthma rarely reveals clinically significant findings, although it may show streaky infiltrates or hyperinflation of the lung fields (Morris and Perkins, 2005). The clinician may also utilize chest x-rays to rule out other pulmonary diseases which can manifest with symptoms of reactive airway disease such as allergic bronchopulmonary aspergillosis or sarcoidosis (Camargo, 2006).

2.8.4.6 Allergy Skin Testing

Allergy skin testing is a useful adjunct in individuals with atopy (Morris and Perkins, 2005). The results help guide indoor allergen mitigation or help diagnose allergic rhinitis symptoms (Morris and Perkins, 2005; Gina report 2007). Allergy immunotherapy may be beneficial in controlling allergic rhinitis and asthma symptoms for some patients (Morris and Perkins, 2005; Gina report 2007).

2.8.4.7 Methacholine- or Histamine-Challenge Testing

Bronchoprovocation testing with either methacholine or histamine is useful when spirometry findings are normal or near normal, especially in patients with intermittent or exercise-induced symptoms (Morris and Perkins, 2005). It also helps to determine if hyperreactive airways are present and a negative test result usually excludes the diagnosis of asthma (Morris and Perkins, 2005). Methacholine is administered in incremental doses up to a maximum dose of 16 $mg.mL^{-1}$, and a 20% decrease in FEV_1, up to 4 $mg.mL^{-1}$ level, is considered a positive test result for the presence of bronchial hyperresponsiveness (Morris and Perkins, 2005). The presence of airflow obstruction with an FEV_1 less than 65-70% at baseline is generally an indication to not perform the test (Morris and Perkins, 2005).

2.9 COMPLICATIONS OF ASTHMA

A serious complication of asthma is status asthmaticus (Haslett *et al*., 2003; The Merck Manual, 2003). Spontaneous pneumothorax is an uncommon but a well-recognized phenomenon (Canaday and Collins, 2004; The Merck Manual, 2003) which may be accompanied by sharp chest pain (The Merck Manual, 2003). Noisy breathing may mask potential physical findings in pneumothorax and pneumomediastinum (Canaday and Collins, 2004). Mediastinal and subcutaneous emphysema due to alveolar rupture and dissection of air along vessels is occasionally observed (Canaday and Collins, 2004; The Merck Manual, 2003), especially in children (Canaday and Collins, 2004). Pneumopericardium, although rare, may occur in younger individuals due to the pericardial layers being loosely apposed as compared to adults (Canaday and Collins, 2004). Bronchiectasis is another rare complication of asthma (Clark *et al*., 1992; The Merck Manual, 2003).

2.10 MANAGEMENT OF ASTHMA

2.10.1 Synopsis of the Medical Management of Asthma

The primary goal of asthma management is the achievement and maintenance of good clinical control (Gina Report, 2007; Gibbon, 2003). In order to achieve this goal it is important to ensure that treatment is directed at minimizing the frequency and severity of symptoms, maintenance of normal lung function and prevention of irreversible changes within the airways (Rabe and Schmidt, 2001). Other objectives of treatment include preventing exacerbation and chronicity of symptoms including night attacks, minimizing the need for emergency hospitalization, maintenance of

baseline (normal) pulmonary function and activity levels and avoidance of adverse treatment effects (The Merck Manual, 2003).

The mainstay of asthma treatment is pharmacological intervention (Gibbon, 2003). Medications to treat asthma can be classified as controllers/preventers or relievers (Rabe and Schmidt, 2001; Risenga *et al*., 2002; Gibbon, 2003; Gina Report, 2007). Sufferers of asthma are required to take their medications on a daily basis even when they experience no symptoms since most medication act by preventing or controlling asthma (Risenga *et al*., 2002). There is no connection between the effects of controller and reliever drugs as their principal mechanism of action is different (Risenga *et al*., 2002).

2.10.1.1 Controller or Preventer Medication

These are medications taken on a daily long-term basis in order to achieve clinical control of asthma primarily through their anti inflammatory effects (Risenga *et al.,* 2002; Gibbon, 2003; Gina Report, 2007). Despite controllers not relieving the symptoms of asthma during an attack, their usefulness lies in their creating a protective shield in the bronchial linings which help to stop or reduce swelling, mucus build-up and muscle tightening in the airways caused by the triggers of asthma (Risenga *et al*., 2002). Since controllers will not work unless used regularly, they, therefore, must be taken on a daily basis (Risenga *et al*., 2002). There are two families of controllers (Risenga *et al.,* 2002) viz. the glucocorticosteroids which include the inhaled and oral forms, and the cromolyns as shown in **Table 2.3**.

Table 2.3 Controller medications prescribed in South Africa for the treatment of asthma*

Drug	Trade names ®	Chemical names
Steroids	**Examples**	**Examples**
Inhaled	Flixotide	Fluticasone
	Inflammide, Pulmicort	Budesonide
	Aerobec, Becotide, Becloforte, Clenil, Ventzone, Viarox	Beclomethasone
Oral		
Syrup	Prelone	Prednisolone
	Celestone, Celestamine	Betamethasone
Tablets		Prednisone
		Prednisolone
Cromolyns		
	Lomudal	Sodium cromoglycate
	Tilade	Nedocromil sodium

*** from Risenga et al. (2002): National Asthma Education Programme**

Table 2.4 The dosages (mcg/day)* of inhaled glucocorticosteroids#

	Low dose (mcg/day)	High dose (mcg/day)
Beclomethasone*	< 1000	1000-2000
Budesonide*	< 800	800-1600
Fluticasone*	< 500	500-1000

*** Adult doses; # from Gibbon (2003): The South African Medicines Formulary.**

A) Glucocorticosteroids

This group of steroids constitutes a wide range of chemicals which are produced both by the body and artificially (Risenga *et al.,* 2002). Steroids are indicated in all patients with severe exacerbations and in the vast majority of patients with moderate exacerbations. The corticosteroids, which are used in the treatment of moderate to severe asthma (Risenga *et al.,* 2002; Gibbon, 2003) are the strongest inhaled controllers (Gina Report, 2007; Risenga *et al.,* 2002; Gibbon, 2003). If the response to the first or second β_2-agonist inhaler treatment is inadequate or incomplete, this would also be an indication for corticosteroids intervention in most patients (Camargo, 2006). Significant improvement in symptoms usually takes one to four weeks to be evident (Gibbon, 2003).

i) Inhaled steroids

According to the Gina Report (2007), inhaled steroids are currently the most the most effective treatment for persistent asthma even though they do not cure asthma. Earlier in 1999, Juniper *et al*. reported that several studies had shown that inhaled steroids were effective in reducing asthma symptoms and improved the lung function and quality of life of asthma suffers. Airway inflammation was controlled (Jeffery *et al*., 1992), airway hyperresponsiveness was decreased (The childhood asthma management program research group, 2000) and mortality rates were reduced (Suissa *et al*., 2000) in asthma sufferers who were on inhaled steroids. However, when these drugs are discontinued, deterioration of clinical control follows within weeks or months in a proportion of patients (Waalkens *et al*., 1993; Jayasiri and Perera, 2005). Although the side-effects are uncommon, they may include hoarse voice or oral thrust, both of which can be prevented by rinsing the mouth with water after inhalation (Gibbon, 2003; Gina Report, 2007). Furthermore, the use of a spacer during inhalation reduces the possibility of side-effects (Risenga *et al.,* 2002).

ii) Short-Term Course of Oral Steroids

This group of drugs is available as syrup or in tablet form (Gibbon, 2003) and is administered for an acute asthma attack for a period of seven to ten days when inhaled steroids have failed (Risenga *et al*., 2002).

iii) Long-Term Course of Oral Steroids

These drugs may be prescribed in asthmatic individuals who have significant symptoms and their asthma is uncontrolled despite high doses of inhaled steroids (Gina Report, 2007; Gibbon, 2003). However, with recent improvements in inhaled steroid therapy, fewer patients are being prescribed the long-term course of oral steroids (Risenga *et al.,* 2002). The therapeutic index of long-term inhaled corticosteroids is considered more favorable than long-term systemic corticosteroids in asthma (Toogood *et al*., 1989; Mash *et al*., 2000).

iv) Injectable Steroids

These steroids can only be administered by a medical doctor or registered nurse and usually only used to treat acute, severe asthma when the patient cannot swallow the oral steroids (Risenga *et al*., 2002).

B) Cromolyns

Cromolyns are stable but extremely insoluble salts which inhibit the release of histamine *in vitro* and prevent bronchial hyperreactivity, while displaying few side effects (Boushey, 2007). The role of cromolyns is limited in the long-term treatment of asthma (Rabe and Schmidt, 2001; Gina Report, 2007) especially in patients with moderate (Risenga *et al*., 2002; Gina Report, 2007) and exercise-induced (Gina Report, 2007) asthma. The anti-inflammatory effect, however, is weak when compared to that inhaled glucocorticosteroids (Cockcroft and Murdock, 1987). This group of drugs may be taken as a powder inhaler, as a nebuliser solution or by aerosol (Risenga *et al*., 2002). When used in aerosols, they effectively inhibit both antigen- and exercise-induced asthma (Boushey, 2007). The side-effects are usually mild and include irritation of the throat, cough, hoarseness of voice, dry mouth, and nausea and vomiting (Rabe and Schmidt, 2001; Risenga *et al*., 2002).

C) Leukotriene receptor antagonists

Leukotriene receptor antagonists are direct antagonist of mediators responsible for airway inflammation in asthma. They are used for prophylaxis of exercise-induced asthma and long-term treatment of asthma as alternative to low doses of inhaled corticosteroids (Morris and Perkins, 2005). Clinical studies have demonstrated that leukotriene modifiers have small and variable bronchodilator effect, reduce symptoms (e.g. cough) (Dicpinigaitis *et al.*, 2002), improve lung function and reduce airway inflammation (Drazen *et al.*, 1999).

2.10.1.2 Reliever Medication

Reliever medication, which are available as inhalants, tablets or syrups (Gibbon, 2003), act quickly to relieve bronchoconstriction and its accompanying acute symptoms (Gibbon, 2003; Gina Report, 2007). Since this group of drugs produces nearly instant relief of symptoms, it is for this reason used as 'first aid' treatment for asthma symptoms or attacks (Risenga *et al*., 2002). Relievers have no effect on the swelling in the airways or the build up of mucous and are used only when the person with asthma has symptoms (Risenga *et al*., 2002; Gina Report 2007). According to Risenga *et al*. (2002), a controller medication should be used if a reliever is used for more than three times a week. The reliever medication prescribed in South Africa according to Risenga *et al*. (2002) is shown in **Table 2.4**.

Table 2.5 Reliever medication prescribed in South Africa for the treatment of asthma

	Trade name ® *	Chemical name*	Dosage[#]
Short-acting bronchodilators *			
Beta-agonists	Abbutamol, Breatheze Salbulin, Venteze Ventolin, Viavent Ipradol Berotec, Fensol Bricanyl	Salbutmol, Hexoprenaline, Fenoterol, Terbutaline	Salbutmol,[*] Aerosol: 100-200 mcg/ 1-2 puffs three to four times daily depending on the symptoms. Oral: two to four mg up to eight mg three to four times daily Fenoterol:[*] Aerosol, 100-200 mg/1-2 puffs one to three daily
Theophyllines	Alcophyllin, Biophyllin Choledyl, Nuelin, Solphyllin Theostat, Vernthol		Oral[*]: Six to eighteen mg/kg/day
Long-acting bronchodilators *			
Beta-agonists	Foradil Serevent	Formoterol, Salmeterol	Formoterol[*]: Aerosol or Powder 12 mg (one puff/ one blister)twice a day Salmeterol[*]: Aerosol or Powder 50 mg (two puffs/ 1 blister) twice a day

Theophyllines	Chronophyllin, Euphyllin Retard Microphyllin, Neulin SA Tabs Theo-Dur, Theoplus, Uni-Dur Uniphyl	Dosages are dependent on the patients symptoms

*** from Risenga *et al*. (2002); # from Gibbon (2003) The South African Medicines Formulary**

A) Inhaled bronchodilators

The primary action of bronchodilators is to decrease muscle tone in both small and large airways in the lungs, thus increasing airflow and ventilation (Gibbon, 2003; Gina Report, 2007). This group of drugs includes the β-adrenergic, methylxanthine and anticholinergic medications (Gibbon, 2003; Camargo, 2006). By far the most effective bronchodilators in the treatment of asthma are the β-agonists (or β_2-adrenoceptoragonists) (Rabe and Schmidt, 2001). Inhaled bronchodilator medication is preferred as it enters directly and rapidly to the airways thus allowing for the administration of smaller doses than those of oral medications. Two types of inhaled bronchodilators are available viz. short-acting and long-acting β-agonists (Gina Report, 2007; Risenga *et al*., 2002; Rabe and Schmidt, 2001). Poor administration technique by patients (Gibbon, 2003; Duarte, 2004), the inability or general disregard of slow inhalation and holding the breath for ten seconds after inhalation by the patient (Gibbon, 2003) may affect the efficacy of the inhaled bronchodilator drugs.

i) Short-acting β_2-agonists

These are medications of choice for the relief of bronchospasm during acute exacerbations of asthma and for the pre-treatment of exercise-induced asthma (Gina Report, 2007; National Guidelines for the Management of Asthma in Adult Asthma at Primary Level, 2002). Although the debate on whether this group of medication needs to be taken on a regular basis or a needed basis has not been resolved (Sears *et al.,* 1991; Spitzer *et al.,* 1992; Page, 1993; Wanner, 1995), The National Guidelines for the Management of Asthma in Adult Asthma at Primary Level (2002) recommends that this group of medication should preferably be used when needed and at its lowest recommended dose and frequency of use (Gina Report, 2007). Butchers *et al*. (1991) have reported that there is some evidence that short-acting β_2-agonists exhibit anti-inflammatory effects although direct proof of this has been difficult to obtain *in vivo* (Rabe and Schmidt, 2001). One of the warning signs of deterioration in asthma control is an increased frequency of use of short-acting

bronchodilators (Crane *et al*., 1989; Sears *et al.,* 1990; Cheung *et al.,* 1992) and this necessitates the need to reassess the treatment protocol (Gina Report, 2007).

There has been some controversy surrounding the potential role of β_2-agonist preparations in asthma mortality (Crane *et al*., 1989; Cheung *et al.,* 1992). The hypothesis is that excessive or regular use of β_2-adrenergic bronchodilators can actually worsen asthma (Cheung *et al.*, 1992) and perhaps contributing to morbidity and mortality (Crane *et al*., 1989). Dennis *et al*. (2000), on the hand, reported that there was no evidence that regular use of inhaled salbutamol 400 µg four-times daily for a year increased the exacerbation rate of asthma. Bronchodilation occurs within five to 15 minutes after inhalation of a short-acting bronchodilator (Gibbon, 2003). The short-acting inhaled β_2-agonist bronchodilator, salbutamol, has duration of action of four to six hours in most patients (Gibbon, 2003).

ii) Long-acting β_2-agonists

These drugs have duration of action of about 12 hours (Risenga *et al*., 2002) and are prescribed for troublesome nocturnal and activity- or exercise-induced symptoms (Risenga *et al*., 2002). They are always used with controller medications and are available as syrups, tablets or capsules (Gina Report, 2007). In South Africa, oral β_2-agonists and theophyllines are the two available types of long-acting β_2-agonists (Risenga *et al*., 2002; Gibbon, 2003).

B) Oral β_2-agonists

These are used in patients where the use of inhaled medication is not possible in situations were slow release preparations may be of value in patients with nocturnal asthma (Gibbon, 2003) even though a higher prevalence of adverse effects has been associated with these drugs (Gina Report 2007; Gibbon, 2003). Side effects such as shakiness, headache, sleeplessness and nervousness have been reported (Risenga *et al*., 2002).

C) Theophyllines

Although theophylline is chiefly a bronchodilator (Gina Report, 2007), anti-inflammatory effects have been reported when it is given in lower doses (Sullivan *et al*., 1994; Barnes, 2003). Despite some evidence suggesting that sustained released of theophylline has little effect as a first line controller (Dahl *et al.,* 2002), it may provide benefit as an add-on therapy in patients who do not achieve control on inhaled glucocorticosteroids alone (Rivington *et al*., 1995; Ukena *et al*., 1997). Recently, this group of medications has been prescribed less frequently due mainly to the

gastrointestinal side-effects such as dyspepsia, indigestion, heartburn, loss of appetite, and nausea and vomiting (Risenga *et al*., 2002). There are two types of theophyllines available in South Africa viz. the short-acting and long-acting types. The short-acting theophyllline medications have a duration of action of four to six hours while the long-acting types have a duration of action of about 12 hours and are started in low doses and taken once or twice a day (Risenga *et al*., 2002; Gibbon, 2003).

2.10.2 ALTERNATE TREATMENTS OF ASTHMA

Complementary and alternative medicine in general has the role of complementing “mainstream medicine by contributing to a common whole, by satisfying a demand not met by orthodoxy or by diversifying the conceptual frameworks of medicine” (Ernst *et al*., 1995). Alternative therapies have become popular with the general public (Eisenberg *et al.,* 1993) and some primary care providers; therefore, these modes of therapies should be closely examined for their efficacy (Reilly, 1983; Eisenberg *et al.,* 1993). Also, patients may desire non-pharmacological therapies to complement their standard pharmacological treatment (Lane, 1991) especially for chronic rather than life-threatening medical conditions (Nielsen *et al*., 1995). Several alternate or complementary treatments may be utilised in the management of asthma. These include hypnosis, herbal remedies, homeopathy, massage therapy, yoga, reflexology, dietary intervention, acupuncture and chiropractic. Although Lehrer *et al.* (2004) states that there is a lack of evidence for the effectiveness of many of these therapies, he is of the opinion that an effective non-pharmacologic alternative or adjunctive treatment of asthma could provide a potentially useful contribution to asthma care. A summary of the studies that have investigated the effectiveness of several alternate treatments of asthma, besides chiropractic or SMT is shown in **Table 2.6**.

Table 2.6 A summary of the studies that have investigated the effectiveness of several alternate treatments of asthma

Reference	Therapy	Sample	Intervention	Results
Maher-Loughnan *et al.* (1962)	Hypnosis	Randomized study of 62 pts consisting of 2 groups.	Group 1: Hypnosis therapy. Group 2: Bronchodilator. Pts were treated for 6 mths with 1 mth observation.	Group 1: ↑ symptomatic improvement; wheezing was ↓ by ½ within 3 mths. Group 2: Little change in symptoms.
Ewer and Stewart (1986)	Hypnosis	39 adults.	6-wk course of hypnosis.	12 pts showed a 74.9% improvement in the degree of bronchial hyper-responsiveness to a methacholine challenge test. PEF rates ↑ by 5.5% and use of bronchodilators ↓ by 26.2%.
Morrison (1989)	Hypnosis	16 chronic asthmatic pts.	Hypnotherapy for 1 year.	↓ in admissions from 44 in the year before starting therapy to 13 in the year after. Hospital stay was ↓ for 13 pts by 249 days; reliance on prednisolone was ↓ in 14 pts. Side effects of the drugs were ↓.
Hackman *et al.* (2000)	Hypnosis	N/A.	Review of hypnosis research.	Hypnosis may be an aid in the treatment of asthma.
Yu and Lee (1976)	Acupuncture	20 pts.	Needle acupuncture performed at 3 sites.	No wheezing and ↓ in symptoms of bronchoconstriction in 5 pts. ↑ in the mean FEV_1 and FVC but not in MMFR.
Fung *et al.* (1986)	Acupuncture	19 children with exercise-induced asthma.	Real and sham acupuncture. FEV_1, FVC, PEF were measured throughout acupuncture and after treadmill exercise.	Mean maximum percentage ↓ in FEV_1, FVC, and PEF were 44.4%, 33.3%, and 49.5% without acupuncture; 23.8%, 15.8%, and 25.9% after real acupuncture; and 32.6%, 26.1%, and 34.3% after sham acupuncture.
Kleijnen *et al.* (1991)	Acupuncture	N/A.	Reviewed 13 trials on the efficacy of acupuncture in the treatment of asthmatic pts.	No study earned more than 72% of the maximum score.
McCarney *et al.* (2003)	Acupuncture	N/A.	Searches in the Cochrane Airways Group Specialized Register, the Cochrane Complementary Medicine Field trials register, the Alternative Medicine Database and reference lists of articles. Contact with the researchers in the field of CAM research.	There was insufficient evidence to make recommendations about the value of acupuncture in asthma Rx.
Zwölfer e*t al.* (1993)	Acupuncture	17 asthmatic pts.	The subjective effectiveness of the Rx was determined by a questionnaire, which was sent to the	Over 70% of the patients reported a significant ↑ in

			patients' homes half the year after starting the acupuncture treatment.	their ailments after 10 wks of Rx as well as half a year after starting acupuncture.
Nagarathna and Nagendra (1985)	Yoga	106 asthmatic pts.	Yoga exercise training for 2 wks, Daily practice for 65 min. Control group of 53 pts with asthma matched for age, sex, type and severity of asthma, who continued to take their usual medications.	There was a significantly greater ↑ asthma attacks and PEF rates in the group who practiced yoga.
Khanam *et al*. (1996)	Yoga	9 pts	Daily yoga.	↓ in heart rate and ↑ in pulmonary ventilation function.
Reilly *et al*. (1994)	Homeopathy	28 pts with allergic asthma.	The pts received either oral homoeopathic or placebo medication. The test Rxs were given as a complement to their unaltered conventional care.	Homeopathic Rx was superior to placebo in visual analogue scores, respiratory function and bronchial reactivity tests.
McCarney *et al.* (1998)	Homeopathy	N/A.	The Cochrane Airways Group Specialized Register of trials; 6 trials with a total of 556 subjects were included.	No trial reported a significant difference on validated symptom scales.
Field *et al.* (1998)	Massage or Relaxation therapy	31 children with asthma aged 6-8 yrs.	Randomly assigned to receive either massage therapy or relaxation therapy: 20 min for 30 days.	Children who received massage therapy showed an ↓ in behavioral anxiety and cortisol levels.
Huntley and Ernst (2000)	Herbal medicines	N/A.	Independent literature searches on Medline, Pubmed, Cochrane Library, and Embase; 17 trials were found.	9 of the 17 trials reported a clinically relevant ↑ in lung function and/or symptom scores.
Petersen *et al.* (1992)	Reflexology	30 pts divided in 2 groups of 15 each.	Group 1: Foot zone therapy. Group 2: Clinical care but without "placebo foot zone therapy". A total of 10 foot zone therapy sessions of 1 hr at intervals of 1 wk.	↓ in consumption of β_2 agonists and ↑ in PEF levels were observed in both groups.
Okamoto *et al.* (2003)	Spa Therapy combined with Dietary Supplement ation with omega-3 fatty Acids	14 asthmatic pts.	Spa therapy and diet of perilla seed oil rich in α linolenic acid, for 8 wks. Generation of LT-C4 by leucocytes and respiratory function were analyzed.	The generation of LT-C4 by leucocytes ↓ for 2, 4 and 8 wks ($p < 0.05$). PEF ↑ significantly for 2, 4, 6 and 8 wks. FVC, FEV_1, FEF_{25}, FEF_{75}, $FEF_{25\text{-}75.75}$ revealed a significant ↑ after 4 and 8 wks of the modified diet ($p < 0.05$).
Graham and Blaiss (2000)	N/A	N/A.	Computer-assisted MEDLINE searches for articles on “CAM” or “herbal therapy” and “asthma” or “atopy.”	Available scientific evidence does not support a role for CAM in the Rx of asthma.

pts = patients; wk= week; wks = weeks; mth = month; mths = months; yrs = years; ↑ = increase/improvement; ↓ = decrease/reduction; Rx = treatment; Rxs = treatments; min = minutes; hr = hour; N/A = Not applicable; FEV_1 = Forced expiratory volume in one second; FVC = Forced vital capacity; FEF_{25} = forced expiratory flow after 25% of expired FVC; FEF_{75} = forced expiratory flow after 75% of expired FVC; $FEF_{25\text{-}75.75}$ = mean expiratory flow during the middle half of the FVC; PEF = Peak expiratory flow; MMFR = Maximal mid-expiratory flow rate; LT-C4 = leukotriene complement 4; CAM = complementary or alternative medicine

Although Maher-Loughlan *et al*. (1962) and, later, Ewer and Stewart (1986) and Morrison (1989) **(Table 2.6)** suggest that hypnosis may be a useful adjunctive therapy in the treatment of asthma, the reported success of hypnotic studies is, however, questionable owing to the lack of matched

control groups and appropriate physiological and psychological measurements (Ewer and Stewart, 1986). The studies have not investigated whether any apparent improvement is a result of a decrease in bronchial responsiveness or simply a decrease in awareness of the degree of bronchoconstriction. The latter possibility could endanger life if it resulted in delay or failure to seek effective treatment in an attack of asthma (Ewer and Stewart, 1986). The very nature of hypnotherapy does not allow for the conduction a double-blind, controlled study (Hackman *et al.*, 2000). Furthermore, investigator (in)experience with hypnosis and the duration of hypnotherapy may also affect the efficacy of hypnosis as a treatment (Hackman *et al.*, (2000).

The literature is ambivalent with the respect to the effectiveness of acupuncture in the treatment of asthma. In 1979, the World Health Organization (WHO) listed 40 diseases for which acupuncture was considered to be beneficial. Diseases of the respiratory tract including asthma and bronchitis were included in that list (Fulder, 1988). Yu and Lee (1976) had reported that acupuncture was useful in alleviating the symptoms of asthma **(Table 2.6)** although there was no control group in their study. Fung *et al*. (1986) **(Table 2.6)** investigated the efficacy of acupuncture in the treatment of asthma and concluded that acupuncture provided better protection against exercise-induced asthma than did (placebo) sham acupuncture ($p < 0.05$). In a study conducted at the Department of Anaesthesia and Intensive Care in the University Hospital of Vienna the findings revealed that over 70% of patients with long-standing asthma reported a significant improvement of their ailments after ten weeks of acupuncture treatment (Zwölfer *et al.,* 1993). On the other hand, Kleijnen *et al*. (1991) **(Table 2.6)** who had earlier reviewed studies on the basis of 18 pre-defined methodological criteria reported that claims that acupuncture is effective in the treatment of asthma are not based on the results of well-performed clinical trials. This is supported by the findings of the members of the Cochrane Airways Group who reviewed all clinical trials that investigated the role of acupuncture (with treatment duration ranging from one to 12 weeks) to control or alleviate the symptoms of asthma. In total, seven trials involving 174 patients were reviewed. All patients continued taking their prescribed asthma medication in conjunction with the acupuncture treatments. The trials reviewed had variable quality and inconsistent results. No significant or clinically relevant effects were found for those patients who received acupuncture treatments (Linde *et al*., 2001). Later, McCarney *et al.* (2003) also concluded that there was insufficient evidence for the use of acupuncture in the treatment of asthma **(Table 2.6)**.

Yoga has been shown to reduce bronchial hyper-responsiveness (Nagarathna and Nagendra, 1985). Khanam *et al*. (1996) reported that asthmatic patients' resting heart rate was significantly decreased following yoga training and there was a significant improvement in pulmonary ventilation

function (probably because of the relaxation of both the voluntary inhalation and exhalation muscle groups) **(Table 2.6)**. The researchers concluded that short-term yoga training for patients should be considered as a self-help tool to complement other forms of treatment.

There is a lack of strong support for the use of homeopathy in the treatment of asthma as reported by McCarney *et al*. (1998) and Linde and Jobst (2000) although, earlier, Reilly *et al*. (1994) did report results which showed that homeopathic treatment was superior to placebo **(Table 2.6)**. Following their study to determine the effect of massage or relaxation therapy on asthmatic children, Field *et al*. (1998) reported that massage therapy is calming and potentially beneficial for asthma **(Table 2.6)**. The beneficial effect of massage therapy could be due possibility that the immune system is influenced by the brain as reported by Vollhardt (1991) in a literature review of clinical trials of similar relaxation interventions. Neuropeptides such as substance P and many types of endorphins are able to modulate leucocytes as these cells have neuropeptide receptors on their surfaces (Payan and Goetzl, 1985; Carr and Blalock, 1986; Ballieux and Heijnen, 1987).

Huntley and Ernst (2000) **(Table 2.6)**, reviewed 17 randomised clinical trials, six of which concerned the use of traditional Chinese herbal medicine and eight described traditional Indian medicine, of which five investigated *Tylophora indica.* Three other randomised trials tested a Japanese Kampo medicine, marijuana, and dried ivy leaf extract. The findings of these authors suggest that herbal remedies may have a role in the management of asthma as 53% of the studies reviewed reported a clinically relevant improvement in lung function and/or symptoms scores. This is in contrast to the report of Graham and Blaiss (2000) who reviewed pertinent abstracts and articles from Medline searches for articles on "complementary/alternative medicine" or "herbal therapy" and "asthma" or "atopy." to ascertain specific complementary/alternative therapies used to treat asthma and the efficacy of these therapies based on the available scientific literature. They concluded that the available scientific evidence does not support a role for complementary/alternative therapies in the treatment of asthma. Huntley and Ernst (2000) did, however, state that the overall quality of the trials they reviewed was poor.

Reflexology (Petersen *et al*., 1992) and spa therapy with dietary supplementation (Okamoto *et al*., 2003) may have a role as adjunctive therapies in the management of asthma **(Table 2.6)**. Further research is, however, needed to determine the usefulness of these therapies in asthmatic patients.

2.10.3 SPINAL MANIPULATION IN THE TREATMENT OF ASTHMA

Nielsen *et al*. (1995) are of the view that SMT is one of the more common complementary therapies utilized by the general public and a significant number of patients with asthma receive chiropractic care. Several practitioners of SMT were of the opinion that SMT has a role in the management of asthmatic patients (Jamison *et al*., 1986). A chiropractor's role as a primary contact physician enables him/her to treat selective cases while referring unresponsive cases to physicians and specialists (Jamison *et al*., 1986). A summary of the studies that have investigated the effect of SMT in the management of asthma is shown in **Table 2.7**.

Table 2.7 A summary of the studies that have investigated the effectiveness of spinal manipulative therapy in the management of asthma

Reference	Intervention	Population/sample	Outcome measures	Procedure	Outcome
Nielsen *et al*. (1995)	SMT to the entire spine.	31 chronic asthmatic patients aged 18-45 years.	FEV_1, FVC, PEF, daily use of inhaled bronchodilators, patient-rated asthma severity, diaries and non-specific bronchial reactivity.	Patients were randomized to receive either active SMT or sham SMT twice weekly for 4 weeks, and then crossed over to the alternative treatment for another 4 weeks.	No statistically significant differences were found between the active and sham chiropractic interventions.
Jamison *et al*. (1986)	SMT and mobilisation of the thoracic spine, STT.	15 male and female asthmatic subjects.	FEV_1, FVC and MMV.	Each patient received 7 treatments over a 5- week period 2 treatments in both the 1st and 2nd weeks and thereafter 1 treatment.	Insufficient evidence to suggest that SMT reduces respiratory obstruction in asthmatics
Gobrin (1997)	SMT – C0 to C2 and T2 to T7.	30 chronic asthmatics.	FEV_1, FVC, and sputum eosinophil level, an asthma questionnaire and an asthma disability index.	Block 1 – objective and subjective measurements. Block 2 - SMT. Block 3 – Disability index completed; spirometry and sputum analysis.	Subjects felt that their asthma had improved. The objective measurements showed significant improvements ($p < 0.05$) in the FEV_1, FVC and sputum eosinophil levels in the subjects. A reduction in their daily medication was also noticed.
Balon *et al*. (1998)	SMT and massage	91 children age 7 to 16 years	Spirometry, quality of life and overall treatment satisfaction	Group 1: SMT. Group: massage and gentle palpation.	No significant spirometry changes. Quality of life improved in both groups but was not statistically significant.
Nilsson and Christiansen (1998)	A retrospective case record-based study of asthmatic sufferers treated by chiropractors.	200 cases.	Questionnaire.	This was carried out to determine the characteristics which correlate with a beneficial outcome following treatment of asthmatic sufferers by a chiropractor.	Some asthmatic sufferers obtained perceived (subjective) benefit following SMT.
Hondras *et al.* (2000)	Search for trials in, e.g. Medline and specialized databases.	Metanalysis of 473 citations, 68 full text articles.	Observed clinical outcomes for at least 2 weeks.	Data was extracted and trial quality was assessed.	Insufficient evidence to support the use of manual therapies for patients with asthma.
Bronfort *et al.* (2001)	SMT or sham SMT with a 1-year follow-up period.	36 mild to moderate asthmatic patients age 6 to 17 years.	Spirometry, forced volume loop, lung volume, plethysmography and nonspecific bronchial challenge with exercise challenge, morning and noon PEF rates, quality of life questionnaire and a diary to record symptoms.	20 chiropractic treatment sessions were scheduled during a 3-month intervention phase. There were 2 groups who received either an SMT or sham SMT in addition to their ongoing medical treatment.	After combining SMT with the medical management for pediatric asthma, the children rated the quality higher and their asthma severity lower. These improvements were maintained at the 1 year follow up. There were no important changes in lung function or hyperresponsiveness.
Leboeuf-Yde *et al.* (2005)	Questionnaire-based study	Multination trial.	Self-administered questionnaire.	Information obtained through questionnaires by chiropractors and patients on return visit within 2 weeks of previous treatment.	Ease of breathing reported by asthmatic patients.

FEV_1 = Forced expiratory volume in one second; FVC = Forced vital capacity; MMV = Maximum voluntary ventilation; SMT = Spinal manipulation; STT = soft tissue therapy; C0 – C2 occiput to cervical vertebra 2; T2 – T7 Thoracic vertebra 2 to thoracic vertebra 7

Evidence for the use of SMT as an intervention in the management of asthma is ambivalent as shown in **Table 2.7.** In addition to these studies, an earlier study on asthmatic patients by Miller (1975) showed that although the improvements in VC, residual volume, total lung capacity and FEV_1 were greater in the SMT group compared to a control group, these were not statistically significant. Hviid (1978) had reported that although SMT was beneficial in improving VC, PEF rate and subjective symptoms in a group of patients with various obstructive pulmonary diseases compared to a control group, no definite conclusions could be reached as the sample size was very small. Jamison *et al*. (1986) and Bronford *et al*. (2001) also reported a reduction of subjective symptoms in asthmatic patients although there was no evidence of an objective improvement **(Table 2.7)**. The mean baseline FEV_1 found by Jamison *et al*. (1986) was 2.38 (range 1.20-5.33) which decreased to 2.22 (range 0.87-5.01) after the first treatment. Bronchospasm following coughing or forced expiration may have contributed to the lack of an objective improvement. This be explained by the possible influence of SMT on the parasympathetic component of the autonomic nervous system (vagus nerves) resulting in bronchoconstriction (Jamison *et al*., 1986). Another explanation for the lack of significant objective findings could be that sub-categories of asthma respond differently to different treatments. For example, asthma that has an allergy-base aetiology may not respond to SMT or other manual interventions (Jamison *et al*., 1986).

In the study of Nielsen *et al*. (1995), the mean baseline FEV_1 was 2.91 which increased slightly to 2.99 during the active SMT phase and dropped slightly to 2.97 during the placebo manipulation phase. One of the reasons why these researchers did not show any statistically significant difference in results between the SMT and the sham manipulation could be due to the technique of the sham intervention itself. The technique involved a patient lying prone on table with a drop-piece for the thoracic and lumbar “manipulations” and lateral recumbent for the cervical “manipulations”. One hand of the practitioner applied gentle pressure over the spinal contact while the other hand thrusted on the drop-piece simultaneously releasing it. Despite the authors reported that this method has been shown to be acceptable placebo intervention (Waagen *et al*., 1986; Brennan *et al*., 1991), it is still possible that the gentle pressure applied over the vertebrae and drop itself could influence the vertebral joint dysfunctions or inaudible cavitations could have also occurred. Furthermore the motion palpation of the spine prior to the sham manipulation could also have had a therapeutic benefit. The inclusion of patients with long-standing moderate asthma who were dependant

on inhaled steroid therapy could have also contributed to the lack of statistical significant findings as it is possible that such patients were unlikely to respond to SMT.

Both Balon *et al*. (1998) and Bronford *et al*. (2001) reported that SMT offered no therapeutic advantage over medical treatment at least in terms of objective findings in paediatric asthmatic patients. With the exception of Nielsen *et al*. (1995) none of the other studies had a homogeneous sample in terms of the diagnostic criteria for the type of asthma according to National Guideline for the Management of Asthma in Adults at Primary Level (2002) and none of the studies reviewed excluded the possible masking effects of the medication i.e. the bronchodilator (Rosner, 2006).

Gobrin's study (1997) demonstrated an improvement in both subjective and objective outcomes in subjects with chronic asthma who received SMT. The entire intervention was subdivided into three block periods. All subjects underwent each of the block periods. Block One consisted of taking the objective measures (FEV_1, FVC, FEV_1 /FVC% and sputum eosinophil levels) and the completion of an asthma questionnaire. Block Two commenced directly after Block One and during this period subjects received SMT to the cervical and mid-thoracic regions. In Block Three, the subjects were randomly divided into two groups with Group One receiving four more chiropractic treatments on a weekly basis while Group Two was only required to attend the Chiropractic Day Clinic four weeks after the completion of Block Two.

In South Africa, one percent of all chiropractic patients with non-musculoskeletal symptoms reported a definite improvement in asthma following chiropractic care compared to a mean of two percent from patients' responses in eight countries (Leboeuf-Yde *et al*., 2005). Nilsson and Christianssen (1998) reported that some asthmatic patients reported subjective improvements following SMT after a record-based study of 200 cases. Hondras *et al*. (2000), on the hand, concluded that there was insufficient support for the use of manual interventions in the treatment of asthma **(Table 2.7)**. Hawk *et al*. (2007) conducted a systematic review to evaluate the effect of chiropractic care (i.e. entire clinical encounter) rather than SMT only in the management of non-musculoskeletal conditions such as asthma, infantile colic and otitis media. They reviewed 47 experimental designs including 14 randomised clinical trials (RCTs), nine systematic reviews and one cohort study. They reported that chiropractic care rather than SMT alone provided benefit to patients with asthma. The findings of this study

suggests that the patient's response may depend on several subjective factors e.g. attitude and beliefs of the practitioner and communication between patient and practitioner.

2.11 CONCLUSION

Despite the popularity of alternative or complementary therapies as adjuncts to pharmacological intervention in the management of asthma (Eisenberg *et al*., 1993), the effectiveness of several of these therapies has not yet been established as shown in **Table 2.5**. Studies investigating the effectiveness of SMT as a useful adjunctive manual intervention have produced conflicting results as reflected in **Table 2.7**. Although encouraging results have been reported by researchers (Hightower *et al*., 1999; Kriel, 2005) on the effect of ribcage mobilisation on chest wall expansion and lung function parameters, no study has yet verified whether similar results would be achieved in asthmatic individuals.

CHAPTER THREE

MATERIALS AND METHODS

3.1 INTRODUCTION

This study was designed as a randomised, controlled, investigative trial, involving three groups of 15 subjects each. Ethical clearance for this study was obtained from the Durban University's Faculty of Health Sciences Ethics Committee **(Ethics Clearance Certificate number: FHSEC033/07, Appendix J)**.

3.2 RESEARCH SITE AND THE SUBJECTS

This study was conducted on-site at the Murchinson District Hospital Asthma Clinic in rural Port Shepstone, in the province of KwaZulu-Natal. The subjects were from the Hospital's Asthma Clinic System which is part of its Out-Patient Department (OPD). The sample size for this study was n = 45 subjects. Financial constraint was the main factor for the relatively small sample size.

3.3 SUBJECT RECRUITMENT

The medical and nursing staff at the Asthma Clinic were approached by the researcher and the nature of the study was explained to them. A document **(Appendix A)** outlining the nature of the study and role of the medical/nursing staff in this study was given to each staff member at the Asthma Clinic. They were given an opportunity to ask questions on the study and the researcher responded accordingly. Those that expressed a willingness to help the researcher in subject recruitment were required to sign an informed consent form **(Appendix B).** The role of these staff members was to introduce any prospective subject to the researcher. A few staff members were also required as translators for the researcher/subject consultation.

3.4 THE CONSULTATION

The consultation with the prospective subjects took place in a small screened-off area of the Asthma Clinic to ensure the subjects' privacy. All prospective subjects were explained the nature of the study by the researcher. Each prospective subject was also given a subject information sheet in Zulu **(Appendix C)** or in English **(Appendix D)**. The researcher answered any questions the prospective subject had asked. Those who expressed a willingness to participate in this study were given an informed consent form **(Appendix E)** to sign. If the subject was an illiterate, a right thumb print was taken and an independent witness also signed the informed consent form.

A case history **(Appendix F)** was taken from each subject, followed by a physical examination **(Appendix G)** (which included a spirometer reading to assess for PEF) and a thoracic spine orthopedic examination **(Appendix H)**. This took place to assess for any conditions such as spinal deformity, influenza, rib fractures, etc. that would exclude the subject from the study and to ascertain whether the subject's diagnosis satisfied the medically defined category of chronic moderate asthma **(Table 2.2)**.

3.5 INCLUSION AND EXCLUSION CRITERIA

3.5.1 Inclusion Criteria

- Only male subjects were considered in this study. This was done to keep the sample homogeneous and furthermore, the measurement of chest wall expansion required the researcher to place the measuring tape directly over the skin of the chest. This required the entire chest to be exposed and the procedure might have been uncomfortable for females.
- Subjects had to be between the ages of 18 to 45 years. Subjects older than 45 years were not included because Brandt (2002) found that little radiographic evidence of osteoarthritis existed in people below the age of 45 years. Any prospective subject under the age of 18 would have required parental consent to participate in this study and for the purpose of this study, an adult was considered as one being over the age of 18 years.
- Subjects on an inhaled short-acting β_2-agonist bronchodilator (salbutamol) were accepted into this study (**Note:** This medication was not prescribed by the researcher. It refers to medication that was previously prescribed by a medical doctor prior to the subjects' participation in this study. The subjects own inhaled bronchodilator was used. Subjects

who were on controller treatment for asthma e.g. corticosteroids in addition to the bronchodilator medication were not excluded).

- Only individuals suffering from chronic moderate asthma were included in this study. The diagnosis of chronic moderate asthma was made by the researcher and the on-site supervising chiropractor based on the criteria of the National Guideline for the Management of Asthma in Adults at Primary Level (2002) **(Table 2.2)**.

3.5.2 Exclusion Criteria

- Subjects were excluded if they had a previous history of rib fractures, dislocations, and sprains of the costochondral, costosternal and interchondral joints.
- Subjects were excluded if they were diagnosed with any cardiac disease e.g. angina or heart failure, etc.
- Any subject who presented with a fever.
- Smokers were excluded from the study.
- Males with gynaecomastia.
- Subjects who presented with any of the following contra-indications to manipulation or mobilisation (Wyatt, 1992; Giles and Singer, 2000):
  - Primary and secondary neoplastic lesions of the spine and/or ribs
  - Obvious advanced spinal deformity e.g. kyphoscoliosis
  - Healing fracture or dislocation
  - Infection e.g. tuberculosis
  - Osteoporosis
  - Inflammatory arthritides e.g. acute rheumatoid arthritis or ankylosing spondylitis
  - Primary and secondary neoplastic lesions of the soft tissue / bony structures of the chest
  - Gross segmental instability of the thoracic spine and/or ribcage
  - Subjects who have any bleeding disorders

  These were excluded on the basis of the case history, physical and orthopaedic examination findings.
- Subjects who experienced an asthma attack during their participation in this study.
- Subjects who were not able to follow the specific instructions issued by the researcher which were crucial to the study e.g. spirometry technique, bronchodilator administration.
- Subjects that were on any of complementary treatments e.g. homeopathy, for a period of two weeks prior to their participation in this study.

- No bronchodilator intake for at least 12 hours prior to the subject's appointment at the hospital (this did not include their controller medication).
- Any subject who refused to sign the informed consent form (or provide a right thumb print).

3.6 RANDOMISATION OF SUBJECTS

An envelope with 45 pieces of paper marked with numbers from 1-45 was used .The subject was asked to take one piece of paper out of the envelope. The subject was assigned to one of three groups depending on the piece of paper he picked from the envelope as follows:

Group A: Short-acting, inhaled β_2-agonist bronchodilator (1-15)

Group B: SMT and ribcage mobilisation (16-30)

Group C: Combination of SMT, ribcage mobilisation and a short-acting, inhaled β_2-agonist bronchodilator (31-45)

3.7 TOOLS AND OUTCOME MEASURES

An objective assessment of changes in the subject during the intervention was required for this study. To this end, two instruments viz. a soft tape measure (Kriel, 2005) and a spirometer (The American Association of Respiratory Care, 2008) were used.

The outcome measures were:

1. Chest wall expansion in centimetres (cm).
2. Spirometry: FEV_1, FVC and FEV_1/FVC%. The IQ Teq spirometer (Jagga House, Cape Town) was utilised in this study **(Appendix I)**.

3.8 INTERVENTIONS

Once the subjects were assigned to one of the three Groups A, B or C they were managed as shown in **Table 3.1**.

Table 3.1 The intervention protocol

Group A (Inhaled, short –acting β_2-agonist bronchodilator)	**Group B** (SMT and ribcage mobilisation)	**Group C** (Combination of (Inhaled, short – acting β_2-agonist bronchodilator, SMT and ribcage mobilisation)
▪ 1. Chest wall expansion and spirometer reading were taken and recorded (baseline). Chest wall expansion measurements were taken circumferentially at the level of the anterior flexure of the axilla, the xiphoid process and halfway between the xiphoid process and umbilicus utilising a soft tape measure in keeping with measurement protocols by Hightower *et al*. (1999). ▪ Corresponding posterior landmarks were found and marked off with a skin pencil. Thus, posteriorly the anterior flexure of the axilla corresponded to the fourth thoracic vertebra; the xiphoid process the eighth thoracic vertebra, while halfway between the xiphoid process and umbilicus corresponded to the twelfth thoracic vertebra (Kriel, 2005). This was done on all subjects. ▪ The subjects had to hold their breath for the duration it took the researcher to measure the chest wall expansion at the three sites. Spirometry Technique (National Guideline for the Management of Asthma in Adults at Primary Level, 2002): The researcher first demonstrated the proper spirometric technique to each subject. ▪ Thereafter each subject was allowed to practice with the instrument until the researcher was satisfied that the subject understood the instructions and was capable of performing the proper spirometric technique. ▪ Three readings were taken within 100m/l or 5% of each other. ▪ The subjects were actively couched and encouraged via verbal instructions, to obtain accurate results. This was the same for all subjects. The highest value was recorded. The subjects were instructed to fully expire and then inspire to their full capacity.	1.Same as Group A.	1.Same as Group A.
2. Two puffs of a self administered inhaled bronchodilator. The subject's own bronchodilator was used (Gibbon, 2003). Inhalation Technique (National Guideline for the Management of Asthma in Adults at Primary Level, 2002): Without a Spacer ▪ The cap was removed from the mouth piece. ▪ The inhaler was shaken well. ▪ While standing or sitting upright, the subject had to maximally exhale. ▪ The mouthpiece of the inhaler was placed	2. Received SMT to the thoracic spine (according to the technique by Bergmann and Peterson (2002) and ribcage mobilisations (according to the technique described by Kriel (2005)). RIbcage mobilisation of Grades 2 to 4 was employed for 10 minutes (Kriel, 2005).	2. Two puffs of a self administered inhaled bronchodilator. The subject's own bronchodilator was used (Gibbon, 2003). Received SMT to the thoracic spine (according to the technique by Bergmann and Peterson (2002) and ribcage mobilisations (according to the technique described by Kriel (2005)). RIbcage mobilisation of Grades 2 to 4 was employed for 10 minutes (Kriel, 2005).

between the lips and the lips were gentle closed around it. ▪ While the subject began to inhale, the canister of the metered dose inhaler was pressed down once to release one puff while the subject had to breath in as deeply as possible. ▪ The breath was held for 5-10 seconds if possible the subject was then asked to breathe out slowly and rest for a few breaths (30-60 seconds). ▪ Steps 2-6 for the second puff were repeated. With a Spacer: ▪ The caps from the inhaler and the spacer were removed. ▪ The inhaler was shaken well. ▪ The mouthpiece of the metered dose inhaler was inserted into the back of the spacer. ▪ The mouthpiece of the spacer was inserted into the mouth and the lips were closed around the mouthpiece. ▪ The canister of the metered dose inhaler was pressed down once to release one puff into the spacer. ▪ 3-6 slow, deep breaths were taken immediately ▪ Steps 4-6 were repeated for each puff prescribed, waiting at least 30 seconds between puffs.		
3. Fifteen minute waiting period (this time was chosen because bronchodilation occurs after five to 15 minutes of administration (Gibbon, 2003)).	3. Same as Group A	3. Same as Group A
4. The subject's chest wall expansion and spirometry reading was retaken and re-recorded.	4. Same as Group A	4. Same as Group A

The subject's regular bronchodilator as prescribed by a medical doctor prior to this study. SMT = Spinal Manipulation

The entire consultation was done under the supervision of an on-site qualified, registered chiropractor. The subject self-administered the inhaled bronchodilator in the presence of a registered medical doctor.

3.9 STATISTICAL CONSIDERATIONS

Data was captured electronically in Excel and imported into the latest version of SPSS (SPSS Inc., Chicago, Ill, USA) for analysis. A p value of <0.05 was considered as statistically significant. The means of the outcome measures of each intervention group were assessed by the use of the paired *t*-test to determine the impact of an intervention protocol on the outcome measure. Baseline outcomes were compared between the three treatment groups using ANOVA with Bonferroni *post-hoc* tests to ensure that the three groups were equivalent in terms of baseline outcomes. The difference between the pre- and post-intervention measurements was calculated and compared between the three groups using repeated measures ANOVA testing. A significant time-group interaction effect would denote a

significant treatment difference between the groups using the Wilk's lambda statistic. All two-way comparisons between groups were done to identify where the difference lies (Esterhuizen, 2007).

3.10 ETHICAL CONSIDERATIONS

- Self-administration (by two groups of subjects i.e. Groups A and C) of a short-acting β-agonist bronchodilator was required during the study. A letter had been provided by a registered pharmacist **(Appendix L)** to the ethics committee to show that no adverse effects would result to these subjects. Furthermore, the self-administration of the bronchodilator was in the presence of a registered medical doctor.
- All the subjects were informed of the nature of the study by the researcher and were also given an information sheet in either English or isiZulu. Each subject signed an informed consent form.
- The entire consultation was supervised on-site by a qualified and registered chiropractor.
- All subjects were thoroughly screened for contra-indications to SMT and/ or rib mobilisations.
- Subjects' names did not appear on any of the data analysis sheets or this dissertation. All subject's names were coded in the data sheets.

CHAPTER FOUR

RESULTS

4.1 SELECTED ANTHROPOMETRIC AND DEMOGRAPHIC DATA

The mean (± SD) of the selected anthropometric data and age of the subjects who participated in this study are shown in **Table 4.1**. The age of the subjects **(Figure 4.1)** ranged from 21 to 45 years. The mass of the subjects ranged from 55 to 102 kg, while the body mass index (BMI) ranged from 16.25 to 32.00 $kg.m^{-2}$. The majority of the subjects in this study were Blacks (*n* = 43) followed by Indians (*n* = 2) and then Coloured (*n* = 1) as shown in **Figure 4.1**. There were no White subjects in this study. Only three of the subjects were employed as shown in **Figure 4.2**.

Table 4.1 Mean ± SD selected anthropometric and age of the subjects (*n* = 45) who participated in this study

Variable	Mean ± SD
Stature (m)	1.77 ± 0.64
Mass (kg)	73.10 ± 10.90
Body Mass Index (BMI) ($kg.m^{-2}$)	23.35 ± 3.82
Age (years)	39.40 ± 4.90

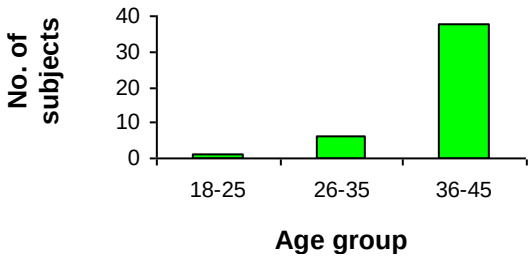

Figure 4.1 The age group of the subjects (*n* = 45) who participated in this study

Figure 4.2 The occupation of the subjects (*n* = 45) who participated in this study

4.2 INTRA-GROUP ANALYSES

4.2.1 Inhaled, Short-acting β_2-Agonist Bronchodilator Group (Group A)

There were no statistically significant changes between pre- and post-intervention in the bronchodilator group as shown in **Table 4.2** although all mean changes in this group were an increase in values between pre- and post-intervention. In terms of lung function values, overall increases were found, but statistical significance was only reached for FEV_1 (p = 0.008; paired t-test).

Table 4.2 Paired *t*-test for comparison of pre- and post-intervention chest wall expansion values in the bronchodilator group (Group A)

		Mean	SD	*p*-value
Pair 1	Axilla pre - Axilla post	-1.800	3.629	0.075
Pair 2	Xiphoid pre – Xiphoid post	-0.267	1.907	0.597
Pair 3	Half-way pre - Half-way post	-0.333	2.257	0.576
Pair 4	Overall mean pre - Overall mean post	-0.799	1.612	0.076

Table 4.3 Paired *t*-test for comparison of pre- and post-intervention lung function values in the bronchodilator group (Group A)

		Mean	SD	*p*-value
Pair 1	FEV_1 pre - FEV_1 post	-0.295	0.373	0.008*
Pair 2	FEV_1/FVC% pre - FEV_1/FVC% post	-1.200	13.995	0.745
Pair 3	FVC pre - FVC post	-0.315	0.611	0.066

***p* < 0.05; paired *t*-test**

4.2.2 Spinal Manipulation and Ribcage Mobilisation (Group B)

There was a statistically significant increase in the mean pre- and post-intervention axillary chest wall expansion (*p* = 0.014; paired t-test) as well as the mean of the half-way measurement (*p* = 0.014; paired *t*-test) and the overall mean chest wall expansion values (*p* = 0.001; paired *t*-test) as shown in **Table 4.4**. There were no statistically significant changes in any of the lung function parameter values in this group **(Table 4.5)**.

Table 4.4 Paired t-test for comparison of pre- and post-intervention chest wall expansion values in the SMT and ribcage mobilisation group (Group B)

		Mean	SD	*p*-value
Pair 1	Axilla pre - Axilla post	-1.200	1.656	0.014*
Pair 2	Xiphoid pre – Xiphoid post	-0.600	1.724	0.199
Pair 3	Half-way pre - Half-way post	-0.933	1.280	0.014*
Pair 4	Overall mean pre - Overall mean post	-0.999	0.873	0.001*

*** *p* < 0.05; paired *t*-test**

Table 4.5 Paired *t*-test for comparison of pre- and post-intervention lung function parameter values in the SMT and ribcage mobilisation group (Group B)

		Mean	SD	*p*-value
Pair 1	FEV_1 pre - FEV_1 post	-0.187	0.205	0.730
Pair 2	FEV_1/FVC% pre - FEV_1/FVC% post	0.552	6.492	0.747
Pair 3	FVC pre - FVC post	-0.045	0.286	0.550

4.2.3 Combination of Spinal manipulation, Ribcage Mobilisation and an Inhaled Short-acting β_2-Agonist Bronchodilator (Group C)

Although all mean changes indicate an increase in chest wall expansion between pre- and post-intervention, the only statistically significant increase in this group was for the half-way measurement (p = 0.018; paired t-test) **(Table 4.6)**. There were no statistically significant changes in any of the lung function parameter values in this group as shown in **Table 4.7**.

Table 4.6 Paired *t*-test for comparison of pre and post-intervention chest wall expansion values in the SMT, ribcage mobilisation and bronchodilator group (Group C)

		Mean	SD	*p*-value
Pair 1	Axilla pre - Axilla post	-0.133	2.588	0.845
Pair 2	Xiphoid pre – Xiphoid post	-0.533	2.588	0.438
Pair 3	Half-way pre - Half-way post	-2.467	3.583	0.018*
Pair 4	Overall mean pre - Overall mean post	-1.040	2.364	0.110

*** $p < 0.05$; paired *t*-test**

Table 4.7 Paired *t*-test for comparison of pre and post-intervention lung function parameter values in the SMT, ribcage mobilisation and bronchodilator group (Group C)

		Mean	SD	*p*-value
Pair 1	FEV_1 pre - FEV_1 post	-0.095	0.327	0.278
Pair 2	FEV_1/FVC% pre - FEV_1/FVC% post	-3.595	8.962	0.143
Pair 3	FVC pre - FVC post	0.094	0.555	0.519

4.3 INTER-GROUP ANALYSES

4.3.1 Axillary, Xiphoid, Half-Way and Overall Mean Chest Wall Expansion

There was no evidence of an intervention effect for these outcomes either in all three groups combined or in each two-way combination of groups ($p > 0.05$; repeated measures ANOVA) as shown in **Tables 4.8**, **4.9**, **4.10** and **4.11**.

Table 4.8 Repeated measures ANOVA for the effect of intervention on axillary chest wall measurements overall and in each intervention group pair

Effect	Statistic	*p*-value
Group overall	Wilk's lambda = 0.937	0.253
Group A *vs*. B	Wilk's lambda = 0.988	0.565
Group A *vs*. C	Wilk's lambda = 0.930	0.159
Group B *vs*. C	Wilk's lambda = 0.939	0.190

***vs*. = versus**

Table 4.9 Repeated measures ANOVA for the effect of intervention on xiphoid chest wall measurements overall and in each intervention group pair

Effect	Statistic	*p*-value
Group overall	Wilk's lambda = 0.995	0.900
Group A *vs*. B	Wilk's lambda = 0.991	0.619
Group A *vs*. C	Wilk's lambda = 0.996	0.650
Group B *vs*. C	Wilk's lambda = 1.000	0.934

***vs*. = versus**

Table 4.10 Repeated measures ANOVA for the effect of intervention on half-way chest wall measurements overall and in each intervention group pair

Effect	Statistic	*p*-value
Group overall	Wilk's lambda = 0.883	0.073
Group A *vs*. B	Wilk's lambda = 0.972	0.378
Group A *vs*. C	Wilk's lambda = 0.880	0.061
Group B *vs*. C	Wilk's lambda = 0.920	0.130

***vs*. = versus**

Table 4.11 Repeated measures ANOVA for the effect of intervention on total chest wall measurements overall and in each intervention group pair

Effect	Statistic	*p*-value
Group overall	Wilk's lambda = 0.996	0.918
Group A *vs*. B	Wilk's lambda = 0.994	0.675
Group A *vs*. C	Wilk's lambda = 0.996	0.774
Group B *vs*. C	Wilk's lambda = 1.000	0.947

***vs*. = versus**

4.3.2 FEV_1, FVC AND FEV_1/FVC%

For FEV_1, the effect in Group A *vs*. B was statistically significant (p = 0.018; repeated measures ANOVA) as shown in **Table 4.12**. There was no evidence of an intervention effect for FVC and FEV_1/FVC% either in all three groups combined or in each two-way combination of groups (p > 0.05; repeated measures ANOVA) as shown in **Tables 4.13** and **4.14**.

Table 4.12 Repeated measures ANOVA for effect of intervention on FEV_1 measurements overall and in each intervention group pair

Effect	Statistic	*p*-value
Group overall	Wilk's lambda = 0.868	0.052
Group A *vs*. B	Wilk's lambda = 0.816	0.018*
Group A *vs*. C	Wilk's lambda = 0.920	0.130
Group B *vs*. C	Wilk's lambda = 0.979	0.448

***** p < 0.05; repeated measures ANOVA**

***vs*. = versus**

Table 4.13 Repeated measures ANOVA for effect of intervention on FVC measurements overall and in each intervention group pair

Effect	Statistic	*p*-value
Group overall	Wilk's lambda = 0.891	0.089
Group A *vs*. B	Wilk's lambda = 0.921	0.133
Group A *vs*. C	Wilk's lambda = 0.883	0.065
Group B *vs*. C	Wilk's lambda = 0.973	0.393

***vs*. = versus**

Table 4.14 Repeated measures ANOVA for effect of intervention on FEV_1/FVC% measurements overall and in each intervention group pair

Effect	Statistic	*p*-value
Group overall	Wilk's lambda = 0.972	0.547
Group A *vs*. B	Wilk's lambda = 0.993	0.663
Group A *vs*. C	Wilk's lambda = 0.989	0.581
Group B *vs*. C	Wilk's lambda = 0.930	0.158

***vs*. = versus**

CHAPTER FIVE

DISCUSSION

5.1 ANTHROPOMETRIC AND DEMOGRAPHIC DATA

The mean (± SD) age of the subjects in this study **(Table 4.1)** indicated that the majority of the subjects in this study were middle-aged males which was in keeping with the inclusion criteria of this study. The age range of the subjects in this study was similar to that of Nielsen *et al*. (1995). The mean (± SD) BMI of the subjects in this study **(Table 4.1)** fell in the "normal" to "overweight" range for adults (World Health Organisation Classification, utilised by Anderson *et al*., 2006). The ethnic profile of the subjects likely reflects the demographics of the surrounding area of Murchison District Hospital **(Figure 5.1)** as it is situated in a semi rural, Black community **(Figure 5.2)**. This hospital serves as an essential health-care service provider mainly to this community situated in the KwaZulu Natal South Coast (Lachman, 2008). The high number of unemployed subjects seen in this study **(Figure 4.2)** is a reflection of the socioeconomic standing of the community. These subjects may not have been able to afford private health-care and, therefore, sought free treatment at the hospital (Lachman, 2008).

Figure 5.1 Entrance to the Murchison District Hospital

Figure 5.2 Semi-rural township surrounding the Murchison District Hospital

5.2 INTRA-GROUP RESULTS

Inhalation of a short-acting β-agonist bronchodilator did not have any significant effect on any of the chest wall expansion measurements **(Table 4.2)**. This indicates that the short-acting bronchodilator had no effect on the movement of the chest wall. Therefore, there would be no "mechanical" effect of the short-acting bronchodilator. This is not a surprising finding since the primary action of bronchodilators is to decrease muscle tone in both small and large airways in the lungs, thus increasing airflow and ventilation (Gibbon, 2003; Gina Report, 2007). According Gibbon (2003), bronchodilation would occur within five to 15 minutes after inhalation of a short-acting bronchodilator. This is supported by the significant increase in FEV_1 following inhalation of the short-acting bronchodilator **(Table 4.3)**.

A significant increase in chest wall expansion (axillary and half-way between the xiphoid and umbilicus) was recorded following SMT and ribcage mobilisation **(Table 4.4)**. This supports the view of Herzog *et al*. (1988), Gal *et al*. (1994), Herzog (2000), Gatterman *et al*. (2001) and Gatterman (2003) who stated that SMT often results in improved flexibility and increased joint mobility. Hightower *et al*. (1999) and Kriel (2005) also reported an increase in chest wall expansion following ribcage mobilisation albeit in elderly adults and healthy, asymptomatic individuals respectively. Although the mobility of the ribs varies considerably in that the first few ribs are more fixed than the others due to the weight of the upper extremities and the

strain of the ribs beneath (Gatterman, 2003), the results of this study suggests that SMT and ribcage mobilisation significantly increases the mobility of this region of the ribcage. Furthermore, increased mobility is seen in the intermediate region of the thoracic cage down to the ninth and tenth ribs. This is supported by Moore and Agur (1996) who state that the first seven ribs are joined to the sternum, the next three have their costal cartilages joined to the rib above them, while the eleventh and twelfth ribs are 'floating' or 'free' ribs as their costal cartilages end in the abdominal muscle wall. The results of this study also support the report of Gibbs (2005) and Stokes (2000) had stated that the motion of the spine may influence the motion of the thoracic cage by virtue of its attachment to the thoracic cage. The increase in chest wall expansion did not translate to an increase in any of the lung function parameters following SMT and ribcage mobilisation **(Table 4.5)**. The pump capacity of the ribcage (Ganong, 1995; Guyton and Hall, 2000) was not influenced by the increase in chest wall expansion following SMT and ribcage mobilisation.

It would have been interesting to determine the effect of the increase in chest wall expansion following SMT and ribcage mobilisation on the volume of the thoracic cavity. McArdle *et al*. (1996) had stated that any change in the volume of the thoracic cavity would result in a corresponding change in lung volume. The lung function parameters are largely influenced by status of the bronchi (i.e. either bronchodilation or bronchoconstriction) and not by an increase in expansion of the chest wall.

The combination of a short-acting β_2-agonist bronchodilator, SMT and ribcage mobilisation resulted in a significant increase in chest wall expansion (at the half-way area between the xiphoid and umbilicus) **(Table 4.6)** but no significant effect on any of the lung function parameters **(Table 4.7)**. The possible influence of SMT on the parasympathetic component of the autonomic nervous system (vagus nerves) resulting in bronchoconstriction (Jamison *et al*., 1986) may have negated the effects of the short-acting bronchodilator **(Table 4.3)**. The increase in chest wall expansion in Groups B and C may have been due to a mechanical response (i.e. stretching of the intercostals muscles and/or costovertebral mobilisation (Kriel, 2005)) or a neural response (via stimulation of the mechanoreceptors (Gandevia and Macefield, 1989; Powers and Howley, 1997)). The expected effect of this on the lung function parameters as hypothesised by Gonzales *et al*. (1999) did not occur.

5.3 INTER-GROUP RESULTS

No subject who received SMT or ribcage mobilisation reported any adverse reactions in keeping with the studies of Nielsen *et al*. (1995), Jamison *et al*. (1986), Balon *et al*. (1998) and Kriel (2005). Although there was no long-term follow-up of these subjects to determine whether any adverse reactions occurred, the findings of this study suggests that these are relatively safe procedures provided the clinician has thoroughly examined the patient and rules out the contra-indications.

In terms of chest wall expansion measurements neither group showed a significant change **(Tables 4.8**, **4.9**, **4.10** and **4.11)**. In terms of lung function, for FEV_1, Group A performed significantly better than Group B but not compared with Group C **(Table 4.12)**. Furthermore, the mean baseline FEV_1 of this study (1.70) was considerably lower than those of the subjects in the studies of Jamison *et al*. (1986) and Nielsen *et al*. (1995). Bronchospasm following forced expiration may have contributed to the lack of an objective improvement especially FVC and FEV_1/FVC% since it is possible that SMT may have influenced the parasympathetic component of the autonomic nervous system (vagus nerves) resulting in bronchoconstriction (Jamison *et al*., 1986) which may have negated the effects of the short-acting bronchodilator **(Table 4.3)**. In 1975, Miller had also failed to demonstrate a significant difference in several lung function parameters in a group which received SMT compared to a control group **(2.10.3)**.

Although great care was taken by the researcher to ensure the each subject who had to self-administer the short-acting inhaled bronchodilator did so according to the correct technique, it is still possible that poor administration by the subjects (Duarte, 2004; Gibbon, 2003), the inability or general disregard of slow inhalation and holding the breath for ten seconds after inhalation by the patient (Gibbon, 2003) may have also affected the efficacy of the inhaled bronchodilator drug in Group C. It is also worth noting that the absence of a response to a single exposure to a bronchodilator does not preclude a beneficial response to maintenance therapy (The Merck Manual, 2003).

Another explanation for the lack of significant objective findings could be that sub-categories of asthma respond differently to different treatments. For example, asthma that has an allergy-base etiology may not respond to SMT or other manual interventions (Jamison *et al*., 1986). The inclusion of patients with long-standing moderate asthma some of whom were

dependant on inhaled steroid therapy could have also contributed to the lack of statistical significant findings as it is possible that such patients were unlikely to respond to SMT (Nielsen *et al*., 1995).

Manipulation of the thoracic spine (and ribcage mobilisation) did not evoke a positive somatovisceral response (as suggested by Nielsen *et al*., 1995, Gobrin, 1997 and Pickar, 2002), at least in the short term, with respect to an effect on the lung function parameters.

While there has been anecdotal and research evidence (Gobrin, 1997; Nilsson and Christiansen, 1998; Bronfort *et al.*, 2001; Leboeuf-Yde *et al.*, 2005) to suggest that SMT may be beneficial as an adjunct to regular pharmacological treatment in chronic asthmatics, the results of this study supports those of Nielsen *et al*. (1995), Jamison *et al*. (1996), Balon *et al*. (1998) and Hondras *et al.* (2000) who reported that SMT may have no benefit as an adjunct to pharmacological intervention in chronic asthmatics **(Table 2.6)**. It was not possible to make any direct comparison of the results of this study and those in **Table 2.6** due to differences in the intervention protocols and other methodological reasons.

It is, however, possible that although the SMT and ribcage mobilisation had no effect on the lung function parameters, at least in the short-term, the overall chiropractic care provided to asthmatic patients in a clinical encounter may still be beneficial as suggested by Hawk *et al*. (2007).

CHAPTER SIX

CONCLUSION AND RECOMMENDATIONS

6.1 CONCLUSION

The primary aim of this study was to determine the short-term effect of:

- An inhaled, short-acting β_2-agonist bronchodilator,
- SMT and ribcage mobilisation and
- Combination of SMT, ribcage mobilisation and an inhaled, short-acting β_2-agonist bronchodilator

on chest wall expansion and lung function in chronic moderate asthmatics.

With regards to the primary aim of this study:

- There were no statistically significant changes between pre- and post-intervention in the short-acting β_2-agonist bronchodilator group with respect to any of the chest wall expansion measurements.
- There was a statistical significant change in FEV_1 between pre- and post-intervention in the short-acting β_2-agonist bronchodilator group.
- There was a statistically significant increase in the mean pre- and post-intervention axillary chest wall expansion as well as the mean of the half-way measurement and the overall mean chest wall expansion value following SMT and ribcage mobilisation.
- There were no statistically significant changes in any of the lung function parameter values following SMT and ribcage mobilisation.
- There was a significant increase for the half-way measurement in chest wall expansion in the combination of SMT, ribcage mobilisation and an inhaled, short-acting β_2-agonist bronchodilator group.
- There were no statistically significant changes in any of the lung function parameter values in the combination of SMT, ribcage mobilisation and an inhaled, short-acting β_2-agonist bronchodilator.
- There was no statistical difference in any of the chest wall expansion measurements between all three groups.

- For FEV_1, the effect in the short-acting β_2-agonist bronchodilator group *vs*. the SMT and ribcage mobilisation group was statistically significant.
- There was no statistical difference in FVC and FEV_1/FVC% parameters between all three groups.

In terms of the specific objectives and associated hypotheses that were set out at the onset of the study:

The Null Hypothesis (H_0) which was set with respect to the short-term effect of an inhaled, short-acting β_2-agonist bronchodilator on chest wall expansion and stated that there would be no significant difference in the chest wall expansion measurements following inhalation of a short acting short-acting β_2-agonist bronchodilator was accepted.

With respect to the short-term effect of the inhaled, short-acting β_2-agonist bronchodilator on FEV_1, the Alternate Hypothesis (H_a) which was set and stated that there would a significant increase in FEV_1 after inhalation of a short acting short-acting β_2-agonist bronchodilator was accepted.

The Null Hypothesis (H_0) which was set with respect to the short-term effect of an inhaled, short-acting β_2-agonist bronchodilator on the lung function parameters FVC and FEV_1/FVC% and stated that there would be no significant difference in these parameters following inhalation of a short acting short-acting β_2-agonist bronchodilator was accepted.

The Null Hypothesis (H_0) which was set with respect to the short-term effect of SMT and ribcage mobilisation on chest wall expansion and stated that there would be no significant difference in the chest wall expansion measurements following SMT and ribcage mobilisation was partially rejected.

The Null Hypothesis (H_0) which was set with respect to the short-term effect of SMT and ribcage mobilisation on the lung function parameters FEV_1, FVC and FEV_1/FVC% and stated that there would be no significant difference in these parameters following SMT and ribcage mobilisation was accepted.

The Null Hypothesis (H_0) which was set with respect to the short-term effect of a combination of SMT, rib cage mobilisation and an inhaled, short-acting β_2-agonist bronchodilator on chest wall expansion and stated that there would be no significant difference in the chest wall expansion measurements following SMT, ribcage mobilisation and inhalation of a short-acting β_2-agonist bronchodilator was accepted.

The Null Hypothesis (H_0) which was set with respect to the short-term effect of a combination of SMT, rib cage mobilisation and an inhaled, short-acting β_2-agonist bronchodilator on the lung function parameters FEV_1, FVC and FEV_1/FVC% and stated that there would be no significant difference in these parameters following SMT, ribcage mobilisation and inhalation of a short-acting β_2-agonist bronchodilator was accepted.

With respect to the chest wall expansion measurements, the Null Hypothesis (H_0) which stated that there would be no significant difference in these measurements between the three groups was accepted.

The Alternate Hypothesis (H_a) which was set and stated that there would a significant increase in FEV_1 after inhalation of a short acting short-acting β_2-agonist bronchodilator as compared to the other two groups was partially accepted.

With respect to the FVC and FEV_1/FVC% parameters, the Null Hypothesis (H_0) which was set and stated that there would be no significant difference in these parameters between the three groups was accepted.

6.2 RECOMMENDATIONS

Recommendations for future studies include the following:

- A similar study to be conducted on mild asthmatics in order to determine whether there would significantly different results compared to moderate asthmatics.
- A larger sample size in order to avoid the possibility of Type II error.
- Multiple intervention intervals over a few weeks with a homogeneous sample in order to determine the effect of multiple interventions as opposed to a single intervention. This would also be a more pragmatic approach.

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Appendix A

DURBAN UNIVERSITY of TECHNOLOGY

Medical/Nursing Staff Information Letter

Dear Sir/Madam

My name is Shekaar Rampersad and I am a Master's student in the Department of Chiropractic, Durban University of Technology. In order to conduct my Master's research (on-site) I require your assistance. Please read this document carefully. If there are any questions or queries, please do not hesitate to ask me.

Title of Research: The Short Term Relative Effectiveness of Two Manual Interventions in the Management of Chronic Moderate Asthma.

Principal Investigators: Shekaar Rampersad (Masters Student)
Dr J. Shaik (Supervisor)

Introduction:
As you are aware, asthma is a common respiratory complaint. Sufferers typically complain of chest pain, tightness of the chest, cough, sputum production, and wheeze. The main treatment of asthma is pharmacological i.e. the use of drugs which are prescribed by medical doctors. One of the most common drugs prescribed is salbutamol which is used as an inhaled bronchodilator. Recently there has been increasing attention given to non-pharmacological (non-drug) adjunct therapies (therapies used in conjunction with drugs) in the management of asthma. Two of these therapies which are grouped under physical therapy modalities include spinal manipulation and ribcage mobilisation. There are research based findings and anecdotal reports that suggest that both of these interventions may be useful as adjunctive therapies in the management of asthma but these findings and /or reports have not yet been shown to be conclusive. Therefore the purpose of this study is to determine the effectiveness of spinal manipulation and rib cage mobilisation as an adjunctive intervention protocols in chronic asthma.

It is important to note that this study, does not aim to replace drug therapy (in asthma) with spinal manipulation and/or rib mobilisation.

Your role in this study:
- To refer and introduce prospective subjects (according to the inclusion criteria below) to the me
- Should the need arise, to act as a translator during the researcher/subjective consultation
- Some subjects will self-administer two doses of inhaled bronchodilator (Salbutamol) in the presence of a medical doctor. Therefore, this study requires a medical doctor to be present during this procedure which will only take about one minute.
- In the unlikely event of a subject experiencing an asthma attack during the research, he will be immediately referred to the medical/nursing staff of the Asthma Clinic for treatment as per the Clinic/Hospital protocol.

The inclusion criteria for subjects:

Only male chronic moderate asthmatic subjects
Between the ages of 18 and 45 years
On inhaled bronchodilator (only Salbutamol)

Criteria for chronic moderate asthma:

Daytime symptoms
Cough
Tight chest
Wheeze
>4 weeks (duration of symptoms)
Night time symptoms (as above)
> 4 months
PEF 60-80 %

PEF = Peak Flow Rate

Risks/ Discomforts:

There are no direct or indirect risks to you as a result of your participation in this study. I have already received permission from the Medical Manager of this hospital to conduct this study at the Asthma Clinic.

Remuneration and costs of the study:

There are no remunerations and costs to you should you agree to assist me in my research

Confidentiality:

Your name will not appear in any of the data sheets, thesis or published article.

Withdrawal from the study:
You may withdraw your participation in this study at any time without any negative repercussions

Persons to contact if I have not answered your questions satisfactorily of if problems arise during this research:

Dr. J. Shaik 031-3732588 (Research supervisor)
Mrs. K. Young 031-373 2704 (Head of Department of Chiropractic, Durban University of Technology)
Prof. N. Gwele 031-373 2102 (Dean Faculty of Health Sciences and Chairperson of the Faculty of Health Sciences Research and Ethics Committee, Durban University of Technology)

I, ……,…… agree to assist Shekaar Rampersad with his research as outlined in this document. I will also sign the attached informed consent form as confirmation of my participation in this study.

Isigaba A

DURBAN UNIVERSITY of TECHNOLOGY

Incwadi Yesaziso Nemininingwane Kuwa bezempilo

Ngiyabingelela

Igama lami ngingu Shekaar Rampersad futhi ngiyisitshudeni esenza iqhuzu le Masters kwi candela le Chiropractic, kwi Durban University of Technology. Ukuze ngikwazi kwenza uphenyo-nzulu lwami (Research) lapho ngaphakathi ngidinga usizo lwakho noma lwenu. Ngicela ufundisise lomqulwana wephepha. Uma kunemibuzo noma izinto ezingaqondakali,ungangabazi ukungibuza .

Isihloko sophenyo-nzulu: Impumelelo Ebonakalayo Ezinhlelweni Ezimbili sezenziwe Ukudambisa Isifo Esiyingozi kodwa Selapheka Sokucinana Kwamaphaphu (Chronic Moderate Asthma)

Abaphenyi-Nzulu Abakhulu: Shekaar Rampersad (Umfundi weqhuzu le Masters)
Dokotela J.Shaik (Ongameleyo)

Isingeniso:

Njengoba wazi ukuthi isifo sokucinana kwesifuba sivamisile lapho ku nokuphazamiseka khona ukuphefumula. Abaphethwe yilesi sifo bavamise ukukhala ngobuhlungu esifubeni ,ukucinana kwe sifuba , ukukhwehlela,ukukhipha izikwhehlela nokugonklozela kwe sifuba. Ukukwelapheka okuphambili kweisifo sokucinana kwesifuba (Asthma) kudinga imithi. Enzulu. ;njengokusebenzisa amadragi akhishwa ngo dokotela bemithi. Elinye lama dragi avamise ukusetshenziswa I Salbutamol esetshenziswa ngokuyihogela ukuvula amaphaphu.

Mumva –nje sekuthanda ukuba kubhekisiswe kakhulu ukusetshenziswa kwe zinye izindlela nangemithi engesiwo ama dragi kanye nokusetshenziswa kwezinye izindlela zokwelapha kanye nama dragi. Ezimbili zalezindlela zokwelapha lesiifo ezihlanganiswe ndawonye ohlelweni lwesimanje oluthinta umzimba uqobo ,zihlanganisa ukuthinta umgogodla kanye nebhokisi lonke lamathambo omzimba. Kukhona imiphumela yophenyo-nzulu etholakale ngemibiko yomunto-qobo eveza ngqo ukuthi zombili lezi zindlela zokwelapha zinga setshenziswa ukwengeza ekwelapheni ukucinana kwesifuba .Kodwa lemiphumela kumbe imibiko beyingaka bekwa njengeyokugcina .Ngakho-ke inhloso ngqangi yalesi sifundo ngukuzama ukuthola ukuthi impumelelo yokusebenzisa loluhlelo loku thinta thinta umgogodla nebokisi lamathambo omzimba njengenye yezindlela zokwe ngeza engasetshenziswa ukwelapha isifo sokucinana kwesifuba esinzima.

Kubalulekile ukuba ubheke futhi ukuthi lesisifundo asiqondene nokugudluza uhlelo lokwelapha I asthma ngemithi(drugs) ngohlelo lokuthintathinta umgogodla nokuqondanisa amathambo ezimbambo.

Iqhaza Lakho –ke kuloluphenyo:

- Ukuba u lethe kimi noma ungazise labo Bantu ngendlela ebekiweyo ngezansi
- Uma isidingo siba khona,weana ube ngumhumushi ngesikhathi ngesikhathi kukhulunywa nalowo muntu

- Abanye balaba bantu bayaye bavele bazifakele bona ngaphandle kokwelekelelwa lomuthi ohogelwayo (Salbutanol) phambi kuka dokotela .Ngakho ke loluphenyo ludinga udokotela abekhona nxa sekwenziwa lokhu okuthatha kuphela cishe umzuzu owodwa.
- Esimweni lapho kunokwenzeka ,kodwa okungeke kwenzeke; ukuthi lowo esifunda ngaye ahlaselwe ukucinana ngaleso sikhathi,uyobe esephuthunyiswa kwabezempilo noma abahlengikazi bohlangothi lwe zifo zokucinana kwezifuba (asthma) ukuze elashwe ngokwe migudu nezindlela ze Kliniki noma yesibhedlela ezibekiwe.

Okubalulekile ngalabo Bantu obalethayo

- Makube ngabesilisa ababambekile noma kancane yilesi sifo sokucinana kwe sifuba
- Ababe phakathi kweminyaka yobudala awu 18 kuya ku 45
- Babe ngabantu abavele besebenzisa lomuthi ohogelwayo ovula amaphaphu (Kuphela i-Salbutanol)

Ukuhlungeka kwe zikhawu zalesi sifo esinzima nesiphakathi nendawo

Izimpawu zasebusuku
Uyakhwehlela
Isifuba esicinene
uyagonklozela
> 4 weeks Ukwenzeka kwezimpawu
Izimpawu zasebusuku
>4 Months
PEF 60 -80 % Ukuhefuzela

PEF = Izinga lokuhefuzela

Ubungozi kanye nokuphatheka kabi

Abukho ubungozi ngqo kumbe thize obuqonde kuwe ngenxa yokuzibandakanya kwakho kuloluphenyo.Besengiyitholile nemvume kumpathi omkhulu walesi sibhedlela ukuba ngenze loluphenyo e kliniki yezokucinana kwezifuba.

Imivuzo nezindleko zalolu phenyo

Awukho kona umvuzo kuwe kanti ngokunjalo azikho izindleko ozongena kuzo ngokuvuma ukuzibandakanya naloluphenyo.

Ukuvikeleka kwakho kwe mfihlo

Igama lakho ngeke nangengozi lavela kunoma yimuphi umbhalo noma amapheshana nemiqingo esabalalisiwe.

Isifiso sokuhoxa kuloluphenyo

Uvumelekile ukuhoxa kuloluphenyo noma nini ngaphandle kokoba kube khona okubi okwenzeka kuwe

Abantu abanokuthintwa

Uma ngingazange ngiyphendule imibuzo yakho ngokugculisa noma kukhona izinkinga ohlangabezana nazo ngesikhathi loluphenyo luqhubeka

U –Dokotela J. Shaik 031 -3732588 (Research Supervisor)
Mrs K Young 031-3732704 (Head of Dept of Chiropractic Durban University of Technology)
Prof.N.Gwele 031-3732102 (Dean Faculty of Health & Sciences and Chairperson Sciences Research and Ethics Committee, Durban University of Technology

Mina… u……..ngiyavuma ukusiza u Shekaar Rampersad ngophenyo lwakhe njengoba kuchaziwe kulencwadi .Ngizoyisayina futhi I fomu elichaziwe ,elifakiwe njengemvume nesifakazisolso sokukuzimbandakanya naloluphenyo .

.

Appendix B

DURBAN UNIVERSITY of TECHNOLOGY

INFORMED CONSENT FORM

(To be completed by Medical/Nursing Staff at the Asthma Clinic, Murchison Hospital)

Date :

Title of research project : The Short-Term Relative Effectiveness of Two Manual Interventions in the Management of Chronic Moderate Asthma

Name of supervisor : Dr J. Shaik (M. Tech. Chiro.; M. Med. Sci. (SM)
Tel : 031 373 2588

Name of research student : Shekaar Rampersad
Tel : 082 849 5595

Please circle the appropriate answer **YES /NO**

1. Have you read the research information sheet?	Yes	No
2. Have you had an opportunity to ask questions regarding this study?	Yes	No
3. Have you received satisfactory answers to your questions?	Yes	No
4. Have you had an opportunity to discuss this study?	Yes	No
5. Have you received enough information about this study?	Yes	No
6. Do you understand the implications of your involvement in this study?	Yes	No
7. Do you understand that you are free to withdraw from this study?	Yes	No
at any time	Yes	No
without having to give any a reason for withdrawing, and	Yes	No
without affecting your future health care.	Yes	No
8. Do you agree to voluntarily participate in this study	Yes	No
9. Who have you spoken to? _____		

Please ensure that the researcher completes each section with you
If you have answered NO to any of the above, please obtain the necessary information before signing

Please print in block letters:

Staff Name:_____ Signature: _____

Witness Name:_____ Signature:_____

Research Student Name:_____ Signature:_____

Ushicilelo B

DURBAN UNIVERSITY of TECHNOLOGY

INCWADI EGUNYAZAYO

Usuku **:**

Isihloko socwaningo **:** Impumelelo Ebonakalayo Ezinhlelweni Ezimbili sezenziwe Ukudambisa Isifo Esiyingozi kodwa Selapheka Sokucinana Kwamaphaphu (Chronic Moderate Asthma)

Igama lika Supervisor **:** Dr J.Shaik (M. Tech. Chiro.; M. Med. Sci. (SM)
: 031-3732588
Igama lomfundi ongumcwaningi **:** Shekaar Rampersad
: 082 849 5595

Uyacelwa ukuba ukhethe impendulo	**Yebo**	**Cha**
1. Ulifundile yini iphepha elinolwazi ngocwaningo?	Yebo	Cha
2. Ube naso yini isikhathi sokubuza imibuzo mayelana nocwaningo?	Yebo	Cha
3. Wanelisekile yini izimpendulo ozitholile emibuzweni yakho?	Yebo	Cha
4. Ube nalo yini ithuba lokuthola kabanzi ngocwaningo?	Yebo	Cha
5. Uyithole yonke imininingwane eyanele ngalolucwaningo?	Yebo	Cha
6. Uyayiqonda imiphumela yokuzimbhandakanya kwakho kulolucwaningo?	Yebo	Cha
7. Uyaqonda ukuthi ukhululekile ukuyeka lolucwaningo?	Yebo	Cha
noma inini	Yebo	Cha
ngaphandle kokunika isizathu sokuyeka	Yebo	Cha
ngaphandle kokubeka impilo yakho ebungozini	Yebo	Cha
8. Uyavuma ukuvolontiya kulolucwaningo?	Yebo	Cha
9. .Ukhulume nobani? ----------		

Uma uphendule ngokuthi cha kokungaphezulu, sicela uthole ulwazi ngaphambi kokusayina.

BHALA NGAMAGAMA AMAKHULU:

Igama :_____ Sayina:_____

gama Witness:_____ Sayina: _____

Igama lomfundi ongumcwaningi:_____ Sayina: _____

Appendix C

DURBAN UNIVERSITY of TECHNOLOGY

Inwadi Yolwazi Ngokwenzekayo Yalowo Olethwayo.

I gama lami ngingu Shekaar Rampersad kanti ngenza izifundo zami zeqhuzu le Masters kwi candela le zokwelashw kwzifo e Durban University of Technology. Ukuze ngikwazi ukwenza uphenyo ngalomkhakha lapho ngidinga usizo lwakho.Ngicela ukuba ufundisise leliphepha ngokucophelela.. Uma kunemibuzo ngicela ungesabi ukuyibuza kimini.

Isihloko Sophenyo: Ukusebenza Kwesikhashana kwe Zindlela ezimbili Ekuwehliseni Isimo Sokucinana kwe Ssifuba Esilula.

Abaphambili Kulolu phenyo: Shekaar Rampersad (Umfundi weqhuzu le Masters)
Dr J.Shaik (Owengamele)

Isingeniso:

Isifo sokucinana komoya esifubeni yinto ejwayelekile lapho umuntu eba nezinhlungu esifubeni, akhwehlele,akhiphe izikhwehlela futhi agonklozele. Ukuselapha okusemqoka kwe Asma ukuba kusetshenziswe imithi yo dokotela. Mumva –nje sekuthanda ukwenyuka ukuthi ke kubhekisiswe ezinye izindlela ezisetshenziswayo kanye nayo lemithi ukukwehlisa I Asthma. Kuyenzeka ukuthi ungakwazi kahle nje ukuphefumula kalula isikhashana emva kokwelashwa ngalezindlela .

Inhloso:

Ngukubona ukuthi uma ngivocavoca umgogodla kanye nokunyakazisa amathambo ezimbamabo kuyasiza yini ukwenza ngcono ukuphefumula uma kwenziwe kanyekanye nesifuthu sikadokotela esejwayelekile.

Ama qembu

Uzobekwa kwelinye lamaqembu amathathu.Kuye ngokuthi ngeliphi iphepha olicoshile.

Lamaqembu ama thathu yilawa:

1. Uzodingeka ukuba uphefumule ka bili esifuthweni sakho esinomuthi
2. Uzobe sewugotshwa amathambo omgogodla kunyakaziswe namathambo ezimbambo zesifuba yimina
3. Uzophinda futhi uhogele ka bili esifuthweni somuthi uphinde ugothwe umgogogodla kunyakaziswe namathambo e ezimbambo.

Qaphela –ke: Uma uzohogela kulesisifutho esinomuthi lokho kuzokwenzeka phambili kuka dokotela wemithi.

Lokhu koku gotswha amathambo omgogodla kanye nesifuba,kona kuzokwenziwa phambi kuka dokotela olapha ngendlela yokuvocavoca amathambo omzima.

Inqubo ezolandelwa:

Uzolethwa kimi umuntu wezempilo noma ngu Mhlengikazi e Kliniki yezifuba e Murchison Hospital Ngizobe sengikutshela ukuthi kumayelana nantoni.

Uzobe sewunikwa ithuba lokubuza imibuzo

Ngiyphendule yonke imibuzo

Uma uvuma ukuzibandakanya kuloluphenyo,uzogcwalisa ifomu lokuthi uyavuma.Uma ungakwazi ukubhala igama lakho ufake isithupha bese linikwa omunye onga hlangene naloluphenyo akusyinele.

Uma ungayisayinanga leyofomu futhi singekho isithupha,ngeke ngukuvumele ukuba yinhlenye yaloluphenyo.

Ngizokubuza imibuzo eminingana mayelana nempilo yakho bese ngikuhlola.

Ngizokucela futhi ukuba ufuthe kakhulu emshinini okala ukuthi ungakanani umoya ongena futhi uphume emaphashin akho.

Uzobe sewucosha ipheshana emvilaphweni.Lelipheshana line nombolo.Uzobe ke sewufakwa eqenjini elithize kuye ngkuthi iyiphi inombolo oyicoshile.

Loluphenyo luzothatha cishe I hora lesikhathi sakho.

Qaphela ukuthi ungahoxa noma nini kuloluphenyo ngaphandle kokuhlukumezeka yimi noma ngabntu base kiliniki Ukwelashwa kwakho ukwejwayelekile lakha e kliniki angeke kuphazamiseke ngoba wenqabe ukuzimbandakanya kuloluphenyo.

Ubungozi/Nokungaphatheki kahle

Ungase uzwe unqanyukelwa umoya ngenkathi kuhlolwa ukuhamba komoya
Kungenzeka uzwe inhlungwanaphakathi nomhlane emva kokuvocwavocwa komgogodla.
Ungase futhi uzwe sakukitaza uma senginyakazisa izimbambo zakho

Umhlomulo kukho konke lokhu:

Uzothola usizo lokuhlolwa
Ungase uzwe kuthanda ukuba lula ukuphefumula kancane emva kwa loluphenyo
Uzothola ithuba lokuhlolwa amaphaphu akho mahala

Inzuzo:

Ngeke ukhokwelwe ngokuzimbandakanya kuloluphenyo

Izindleko:

Ngeke futhi okhoke lutho ngokuzimbandakanya kuloluphenyo

Ukulimala ngenxa yaloluphenyo:

Ungase uzizwe unqanyukelwa umoya
Owezokwelapha ngalendlela uzokwelapha uma kwenzeka uzwa izinhlungu maphakathi nogogodla

Kokungejwayelekile ukuba umuntu asukwe yi asthma kodwa ke uyothunyelwa ekiliniki ye asthma ukuba welashwe.

Izimfihlo:

Igama lakho ngeke nangengozi livezwe kunoma yini noma ibhuku okungenzeka ngilibhale.

Izathu ezingenza uyekiswe ukuba yinxenye yaloluphenyo

Uma unesifo esiyingozi njenge TB,umdlavuza,isifo senhliziyo nokopha.
Amathambo akho athabile kakhulu
Une mfiva
Udla imithi kadokotela ungaka tshelwa ukuthi usungakwenza loko
Uma ungumuntu obhemayo
Uma wake walimala maphakathi namathambo ezimbambo
Uma lukhona olunye uhlobo lokwelashelwa iasthma ngaphandle kolwemithis nokwelashwa okujwayelekile.
Uma ungayisayinanga ifomu yokuvuma.
Uma uqalwe I asma ngesikhathi kwenziwa uphenyo.

Abantu abanokuthintwa

Uma ngingazange ngiyphendule imibuzo yakho ngokugculisa noma kukhona izinkinga ohlangabezana nazo ngesikhathi loluphenyo luqhubeka

U –Dokotela J. Shaik 031-3732588 (Research Supervisor)
Mrs K Young 031-3732704 (Head of Dept of Chiropractic Durban University of Technology)
Prof.N.Gwele 031-3732102 (Dean Faculty of Health & Sciences and Chairperson of the Faculty of Sciences Research and Ethics Committee, Durban University of Technology

Mina……...ngiyavuma ukusiza u Shekaar Rampersad ngophenyo lwakhe njengoba kuchaziwe kulencwadi .Ngizoyisayina futhi I fomu elichaziwe ,elifakiwe njengemvume nesifakazisolso sokukuzimbandakanya naloluphenyo .

Appendix D

DURBAN UNIVERSITY of TECHNOLOGY

Letter of Information to the Subject

Dear Sir/Madam

My name is Shekaar Rampersad and I am a Master's student in the Department of Chiropractic, Durban University of Technology. In order to do my Master's research (on-site) I need your help in this study. Please read this paper carefully. If there are any questions or queries, please do not be afraid to ask me.

Title of Research: The Short Term Relative Effectiveness of Two Manual Interventions in the Management of Chronic Moderate Asthma.

Principal Investigators: Shekaar Rampersad (Masters Student)
Dr J. Shaik (Supervisor)

Introduction:
Asthma is a common chest problem where people have chest pain, tightness of the chest, cough, phlegm and wheeze. The main treatment of asthma is the use of medicines which are prescribed by medical doctors. Recently there has been increasing attention given to treatments which are used together with these drugs in the management of asthma. It is possible that you may actually breathe easier for a short while after these therapies.

Purpose:
To see if spinal manipulation (popping of the joints of your mid-back) and rib cage mobilisation (movement of your ribs by me) help in improving your breathing if used with the normal pump medicine prescribed by your doctor.

Grouping:
You will be put into one of 3 groups. This depends on the number of the piece of paper you pick. The three groups are:

1) You will be required to breath-in 2 puffs of your pump medicine only
2) You will get spinal manipulation ("popping of the joints of your midback") and ribcage mobilisation ("movement of your ribs by me")
3) You will need to breath-in 2 puffs of your pump medication and then you will get spinal manipulation ("popping of the joints of your mid-back") and ribcage mobilisation ("movement of your ribs by me")

Note: If you have to take the 2 puffs of your pump medication, then this will be done in front of a medical doctor. The popping of the joints of your mid-back and movement of your ribs by me will be done in front of doctor who specializes in these treatments (a chiropractor)

Procedure:
You will be introduced to me by the Medical/Nursing staff at the Asthma Clinic, Murchison Hospital
I will tell you what this study is all about in detail
You will be given a chance to ask any questions or queries
I will answer all your questions or queries
If you agree to take part in this study, you will have to sign an informed consent form. If you cannot write or sign your name, a thumb print will be taken from you and someone who is not part of this study will sign the form on your behalf. If you do not sign this form or there is no thumb print, I cannot allow you to be part of this study
I will ask you many questions on your medical history and then examine you. I will also ask you to blow as hard as you can into a device which measures how much air enters and leaves your lungs.

You will pick a piece of paper from an envelope. This piece of paper is marked with a number. You will be put into a group depending on which number you pick.
This study will take up about 1 hour of your time.

Please note that you may withdraw from this study at any time without any harm towards you either by me or the Asthma Clinic Staff. Your normal treatment at the Asthma Clinic will not be affected should you not want to take part in this study.

Risks/Discomforts:
You may become breathless on airflow testing
You may have slight mid-back pain after spinal manipulation ("popping of the joints of your midback")
You may feel slightly ticklish when I am doing the ribcage mobilisation ("movement of your ribs by me")

Benefits:
You will receive a medical check-up
You may experience an ease of breathing for a short while after taking part in this study
You will receive a free test of your lungs

Remuneration:
You will not be given any payment for taking part in this study

Costs:
You will not have to pay anything to take part in this study

Research related injury:
You may become breathless for a short while after the testing of your lungs
A qualified, chiropractor will attend to you should you have any mid-back pain
In the unlikely event of you experiencing an asthma attack during this study, you will be immediately sent to the medical/nursing staff of the Asthma Clinic for treatment

Confidentiality:
Your name will not appear on the results of this study or in anything (book) that I may write.

Reasons why you may be taken out of this study without your consent:
You have a serious medical illness e.g. TB or cancer, heart disease, bleeding conditions
Your bones are too weak (osteoporosis)
You have a fever
You take your medication before you are asked to do so
You are a smoker
If you have any major injury to the mid-back or the ribcage area
If you are on any other form of treatment for your asthma (besides your usual medication) e.g. homeopathy
You do not sign the informed consent form (or your thumb print is missing)
You have an asthma attack while you are taking part in this study

Persons to contact if I have not answered your questions satisfactorily of if problems arise during this research:

Dr. J. Shaik 031-3732588 (Research supervisor)
Mrs. K. Young 031-373 2704 (Head of Department of Chiropractic, Durban University of Technology)
Prof. N. Gwele 031-373 2102 (Dean Faculty of Health Sciences and Chairperson of the Faculty of Health Sciences Research, Durban University of Technology)

I, ……..……..... agree to voluntarily participate in Shekaar Rampersad's study as outlined in this document. I will also sign the attached informed consent form as confirmation of my participation in this study.

Appendix E

DURBAN UNIVERSITY of TECHNOLOGY

INFORMED CONSENT FORM

(To be completed by the subject)

Date **:**

Title of research project **:** The Short Term Relative Effectiveness of Two Manual Interventions in the Management of Chronic Moderate Asthma

Name of supervisor **:** Dr J. Shaik (M. Tech. Chiro.; M. Med. Sci. (SM)
Tel **:** 031 373 2588

Name of research student **:** Shekaar Rampersad
Tel **:** 082 849 5595

Please circle the appropriate answer **YES /NO**

1. Have you read the research information sheet? Yes No
2. Have you had an opportunity to ask questions regarding this study? Yes No
3. Have you received satisfactory answers to your questions? Yes No
4. Have you had an opportunity to discuss this study? Yes No
5. Have you received enough information about this study? Yes No
6. Do you understand the implications of your involvement in this study? Yes No
7. Do you understand that you are free to withdraw from this study? Yes No
   at any time Yes No
   without having to give any a reason for withdrawing, and Yes No
   without affecting your future health care. Yes No
8. Do you agree to voluntarily participate in this study Yes No
9. Who have you spoken to? _____

Please ensure that the researcher completes each section with you
If you have answered NO to any of the above, please obtain the necessary information before signing

Please print in block letters:

Patient /Subject Name:_____ Signature: _____

Witness Name:_____ Signature:_____

Research Student Name:_____ Signature:_____

Ushicilelo E

DURBAN UNIVERSITY of TECHNOLOGY

INCWADI EGUNYAZAYO

Usuku **:**

Isihloko socwaningo **:** Impumelelo Ebonakalayo Ezinhlelweni Ezimbili sezenziwe Ukudambisa Isifo Esiyingozi kodwa Selapheka Sokucinana Kwamaphaphu (Chronic Moderate Asthma)

Igama lika Supervisor **:** Dr J.Shaik (M. Tech. Chiro.; M. Med. Sci. (SM)
: 031-3732588
Igama lomfundi ongumcwaningi **:** Shekaar Rampersad
: 082 849 5595

Uyacelwa ukuba ukhethe impendulo	**Yebo**	**Cha**
1. Ulifundile yini iphepha elinolwazi ngocwaningo?	Yebo	Cha
2. Ube naso yini isikhathi sokubuza imibuzo mayelana nocwaningo?	Yebo	Cha
3. Wanelisekile yini izimpendulo ozitholile emibuzweni yakho?	Yebo	Cha
4. Ube nalo yini ithuba lokuthola kabanzi ngocwaningo?	Yebo	Cha
5. Uyithole yonke imininingwane eyanele ngalolucwaningo?	Yebo	Cha
6. Uyayiqonda imiphumela yokuzimbhandakanya kwakho kulolucwaningo?	Yebo	Cha
7. Uyaqonda ukuthi ukhululekile ukuyeka lolucwaningo?	Yebo	Cha
noma inini	Yebo	Cha
ngaphandle kokunika isizathu sokuyeka	Yebo	Cha
ngaphandle kokubeka impilo yakho ebungozini	Yebo	Cha
8. Uyavuma ukuvolontiya kulolucwaningo?	Yebo	Cha
9. .Ukhulume nobani? ----------		

Uma uphendule ngokuthi cha kokungaphezulu, sicela uthole ulwazi ngaphambi kokusayina.

BHALA NGAMAGAMA AMAKHULU:

Igama :_____ Sayina:_____

gama Witness:_____ Sayina: _____

Igama lomfundi ongumcwaningi:_____ Sayina: _____

Appendix F

DURBAN UNIVERSITY OF TECHNOLOGY
CHIROPRACTIC DAY CLINIC
CASE HISTORY

Patient: _____ Date: _____

File # : _____ Age: _____

Sex : _____ Occupation: _____

Intern : _____ Signature _____

FOR CLINICIANS USE ONLY:

Initial visit

Clinician: Signature :

Case History:

Examination:

Previous: Current:

X-Ray Studies:

Previous: Current:

Clinical Path. lab:

Previous: Current:

CASE STATUS:

PTT:	Signature:	Date:

CONDITIONAL:

Reason for Conditional:

Signature: Date:

Conditions met in Visit No:	Signed into PTT:	Date:
Case Summary signed off:		Date:

Intern's Case History:

1. **Source of History:**

2. **Chief Complaint : (patient's own words):**

3. **Present Illness:**

	Complaint 1	**Complaint 2**
< Location < Onset : Initial: Recent: < Cause: < Duration < Frequency < Pain (Character) < Progression < Aggravating Factors < Relieving Factors < Associated S & S < Previous Occurrences < Past Treatment < Outcome:		

4. **Other Complaints:**

5. **Past Medical History:**

< General Health Status

< Childhood Illnesses

< Adult Illnesses

< Psychiatric Illnesses

< Accidents/Injuries

< Surgery

< Hospitalizations

6. Current health status and life-style:

< Allergies

< Immunizations

< Screening Tests incl. x-rays

< Environmental Hazards (Home, School, Work)

< Exercise and Leisure

< Sleep Patterns

< Diet

< Current Medication
Analgesics/week:

< Tobacco

< Alcohol

< Social Drugs

7. Immediate Family Medical History:

< Age
< Health
< Cause of Death
< DM
< Heart Disease
< TB
< Stroke
< Kidney Disease
< CA
< Arthritis
< Anaemia
< Headaches
< Thyroid Disease
< Epilepsy
< Mental Illness
< Alcoholism
< Drug Addiction
< Other

8. Psychosocial history:

< Home Situation and daily life

< Important experiences

< Religious Beliefs

9. Review of Systems:

< General

< Skin

< Head

< Eyes

< Ears

< Nose/Sinuses

< Mouth/Throat

< Neck

< Breasts

< Respiratory

< Cardiac

< Gastro-intestinal

< Urinary

< Genital

< Vascular

< Musculoskeletal

< Neurologic

< Haematologic

< Endocrine

< Psychiatric

Appendix G

Durban University of Technology

PHYSICAL EXAMINATION: SENIOR

Patient Name : _____ **File no :**_____ **Date :**_____

Student : **Signature :**

VITALS:

Pulse rate:			Respiratory rate:	
Blood pressure:	R	L	Medication if hypertensive:	
Temperature:			Height:	
Weight:	Any recent change? Y / N		If Yes: How much gain/loss	Over what period

GENERAL EXAMINATION:

General Impression		
Skin		
Jaundice		
Pallor		
Clubbing		
Cyanosis (Central/Peripheral)		
Oedema		
Lymph nodes	Head and neck	
	Axillary	
	Epitrochlear	
	Inguinal	
Pulses		
Urinalysis		

SYSTEM SPECIFIC EXAMINATION:

CARDIOVASCULAR EXAMINATION

RESPIRATORY EXAMINATION

ABDOMINAL EXAMINATION

NEUROLOGICAL EXAMINATION

COMMENTS

Clinician: **Signature :**

Appendix H

DURBAN UNIVERSITY of TECHNOLOGY

THORACIC SPINE REGIONAL EXAMINATION

Patient:_____ File:_____ Date:_____

Intern:_____ Signature:_____

Clinician: _____ Signature:_____

STANDING:

Posture (incl. L/S & C/S)
Muscle tone

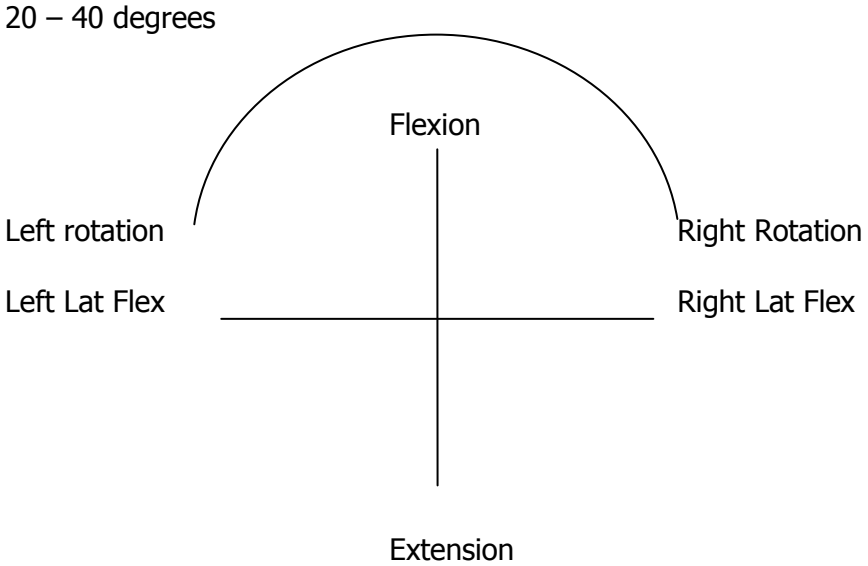
Skyline view – Scoliosis
Spinous Percussion
Breathing (quality, rate, rhythm, effort)
Deep Inspiration

Scars
Chest deformity

(pigeon, funnel, barrel)

RANGE OF MOTION:

Forward Flexion	20 – 45 degrees (15cm from floor)
Extention	25 – 45 degrees
L/R Rotation	35 – 50 degrees
L/R Lat Flex	20 – 40 degrees



RESISTED ISOMETRIC MOVEMENTS: (in neutral)

Forward Flexion
L/R Rotation
Extension
L/R Lateral Flexion

SEATED:

Palpate Auxillary Lymph Nodes
Palpate Ant/Post Chest Wall
Costo vertebral Expansion (3 – 7cm diff. at 4th intercostal space)
Slump Test (Dural Stretch Test)

SUPINE:

Rib Motion (Costo Chondral joints)
Soto Hall Test (#, Sprains)
SLR
Palpate abdomen

PRONE:

Passive Scapular Approximation
Facet Joint Challenge
Vertebral Pressure (P-A central unilateral, transverse)
Active myofascial trigger points:

	Latent	Active	Radiation Pattern		Latent	Active	Radiation Pattern
Rhomboid Major				Rhomboid Minor			
Lower Trapezius				Spinalis Thoracic			
Serratus Posterior				Serratus Superior			
Pectoralis Major				Pectoralis Minor			
Quadratus Lumborum							

COMMENTS:_____

NEUROLOGICAL EXAMINATION:

	DERMATOMES											
	T 1	T 2	T 3	T 4	T 5	T 6	T 7	T 8	T 9	T 10	T 11	T 12
Left												
Right												

Basic LOWER LIMB neuro:

Myotomes	
Dermatomes	
Reflexes	

KEMP'S TEST:

MOTION PALPATION:

			Right	Left
Thoracic Spine				
Ribs	Calliper (Costo-transverse joints)			
	Bucket Handle	Opening		
		Closing		
Lumbar Spine				
Cervical Spine				

BASIC EXAM	History	ROM	Neuro/Ortho
LUMBAR			
CERVICAL			

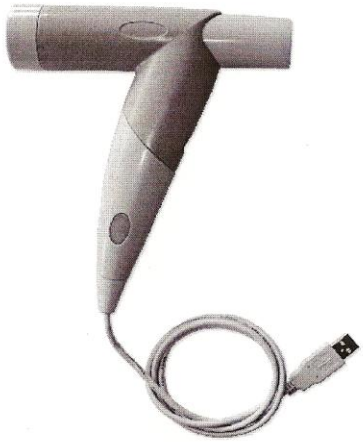
Appendix I

IQ TeQ
MEDICAL

SPIROMETER

Spirometry is the key to diagnosing patients with lung diseases. Primary care physicians need Spirometry to judge responses to therapy and to detect lung diseases in the early stages where treatment is more effective, and to accurately prescribe medication.

The National Lung Health Education Program (NLHEP) recommends Spirometry for all smokers over 20 pack years, and for anyone with coughs, dyspnoea, excess mucus or wheeze.



BENEFITS AND ADVANTAGES OF SPIROMETRY

To Determine the -

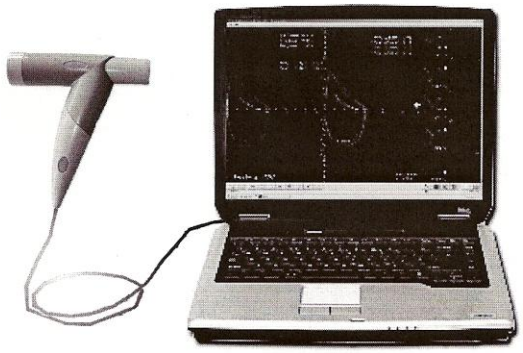
- **Presence** of lung disease or abnormality
- **Extent of abnormalities**
- **Extent of impairment**
- **Progression** of lung disease
- **Nature of the physiologic disturbance**
- **Course of therapy**

*It is time for all **primary care physicians** to equip themselves with a Spirometer that can result in the discovery of important prognostic information for patients, and can guide beneficial therapy in the **primary care setting**.*

(Extract from 'Spirometry made Simple' by Professor Thomas L. Petty, chairman of the NLHEP)

FEATURES

- ❑ Meets all international standards [ATS / ERS]
- ❑ Unique Test procedure quality control
- ❑ Full featured Spirometer with high resolution
- ❑ Easy to use with technique assessment
- ❑ USB port connection to PC
- ❑ Small Portable and lightweight
- ❑ Print Preview
- ❑ Comprehensive Print-copy, *see overleaf*
- ❑ Software enabled for digital data transmission and storage
- ❑ Competitively priced



'TOP OF THE RANGE' FUNCTIONALITY

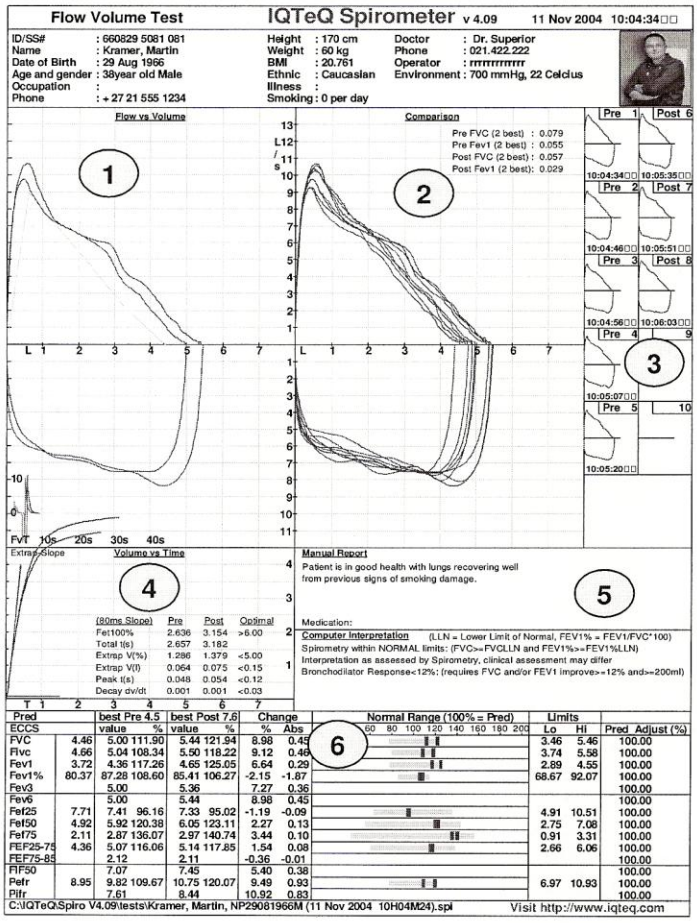
- ❑ Inspiration & Expiration Flow & Volume
- ❑ Flow/Volume & Volume/Time graphs
- ❑ Multiple graph overlays
- ❑ Pre & Post Bronchodilator
- ❑ Diagnostics with Rationale included
- ❑ Real-time test Quality Control assessment

CONTACT IQ TEQ

IQ TeQ MEDICAL

Comprehensive Print Copy with Emphasis on Test Quality Control

The IQ TeQ Diagnostic Spirometer for Doctors and Industry complies with the American Thoracic Society (ATS) recommendations. The system includes the Test Procedure, Test Quality Control, Diagnostic and Print Copy recommendations by the ATS and the American College of Occupational and Environmental Medicine (ACOEM)



- 1 • Pre and Post Bronchodilator Graphs superimposed
  • Automatic best test selection (FVC+FEV1)
  • Predicted graph for easy reference and comparison
- 2 • All selected tests superimposed for repeatability and quality control
  • ATS best test selection criteria displayed
- 3 • Micro display of all tests for easy sequence and selection analysis
  • Time of test displayed
  • Database storage and retrieval
- 4 • Volume /Time graphs with start and end of test criteria for easy quality control assessment (ATS 1994)
- 5 • Comprehensive computer interpretation with rationale for informed decision and assessment of validity
  • Interpretation based on ATS and ACOEM requirements
- 6 • Selection of Reference values
  • Bar Chart display of results with normal ranges for easy assessment

System Description

The IQTeQ Spirometer is a diagnostic Spirometer system for the measurement, recording and assessment of the Flow/ Volume and Volume/Time parameters.

Pneumotach

Differential pressure Laminar flow resistance element. Flow resistance <2kPa at 12 L/sec.

Software Program

Supplied on CD-ROM.

Technical Data

Size: 140x150x45mm (HxLxW) , 200g. Safety: Quality controlled to ISO9001. PC interface: USB (Universal Serial Bus) Resolution and Sample Rate: 12bit 335samples/s. Power Supply: USB bus powered. Calibration: 3Liter Syringe, ATS requirement.

IQ TeQ Spirometer Specifications

Accuracy: Complies with ATS 1994 and ATS 2005 Standards. Range: Flow, Expiratory and Inspiratory 16liters/sec. Volume, 10liters BTPS. Parameters Measured: FVC, FEV1, FEV3, FEV6, FEV1/FVC, FEV1/FEV6, PEFR, FEF25%, FEF50%, FEF75%, FEF25-75, FEF75-85, FIVC, PIFR, FIV0.5, FIV1, FIV3, FIVI/FIVC, FIF50%, , FET100%, SVC Extrapolated V%, and V(L), Peak rise time. Tests Performed: FVC, FIVC, PRE/POST Bronchodilator, Flow/Volume Loop, Volume/Time graph, SVC. Predicted Normal/Reference Equations: Crapo, ECCS, Cherniak, Morris, Knudson1983, Hsu, Schoenberg, HANES III 1999 (Hankinson), Polgar 79.

Caution: Federal Law restricts this device to sale by, or on the order of, a physician.

The IQTeQ Spirometer includes:

The Flow handle and Pneumotach unit, Antibacterial Filter, USB cable, Nose Clip, disposable mouthpieces, Software installed on CD-ROM, Manual.

Calibration Syringe: Optional Extra.

PC Requirements (Minimum):

Pentium II with USB port,
128mb Ram,
Windows 98 Second Edition,
CD-ROM drive, mouse, and
800x600 screen resolution.

[Windows is a registered trademark of Microsoft Corporation.]

Due to continued development and product enhancement, specifications are subject to change without notice.

Appendix J

DURBAN UNIVERSITY of TECHNOLOGY

Faculty of Health Sciences

ETHICS CLEARANCE CERTIFICATE

Student Name	**Mr SR Rampersad**	**Student No**	**20103609**
Ethics Reference Number	**FHSEC 033/07**	**Date of FRC Approval**	**29/10/07**
Research Title:	**The Short Term Relative Effectiveness Of Two Manual Interventions In The Management Of Chronic Moderate Asthma**		

In terms of the ethical considerations for the conduct of research in the Faculty of Health Sciences, Durban University of Technology, this proposal meets with Institutional requirements and confirms the following ethical obligations:

1. The researcher has read and understood the research ethics policy and procedures as endorsed by the Durban University of Technology, has sufficiently answered all questions pertaining to ethics in the DUT 186 and agrees to comply with them.
2. The researcher will report any serious adverse events pertaining to the research to the Faculty of Health Sciences Research Ethics Committee.
3. The researcher will submit any major additions or changes to the research proposal after approval has been granted to the Faculty of Health Sciences Research Committee for consideration.
4. The researcher, with the supervisor and co-researchers will take full responsibility in ensuring that the protocol is adhered to.
5. ***The following section must be completed if the research involves human participants:***

	YES	NO	N/A
❖ Provision has been made to obtain informed consent of the participants	✓		
❖ Potential psychological and physical risks have been considered and minimised	✓		
❖ Provision has been made to avoid undue intrusion with regard to participants and community	✓		
❖ Rights of participants will be safe-guarded in relation to: - Measures for the protection of anonymity and the maintenance of Confidentiality.	✓		
- Access to research information and findings.	✓		
- Termination of involvement without compromise	✓		
- Misleading promises regarding benefits of the research	✓		

SIGNATURE OF STUDENT/RESEARCHER — DATE: 05/06/08

SIGNATURE OF SUPERVISOR/S — DATE: 05/06/08

SIGNATURE OF HEAD OF DEPARTMENT — DATE: 05/06/08

SIGNATURE: CHAIRPERSON OF RESEARCH ETHICS COMMITTEE — DATE: 10/06/08.

Appendix K

2/06/07

To whom it may concern

SALBUTAMOL INHALATION IN CHRONIC ASTHMATIC PATIENTS

*Salbutamol is a short acting selective beta2-adrenoceptor agonist. It is indicated in the treatment of asthma and other conditions associated with reversible airways obstruction. The adult dosage of a Salbutamol inhaler is 100-200ug (1-2 puffs) as required up to 3-4 times daily.

For the purpose of research, administering 2 puffs in a chronic asthmatic without an asthma attack has not shown to cause any adverse effects.

Yours sincerely

I.R Whitfield
B.Pharm (Rhodes)
Reg # 4135

*Extracted from the South African Medicines Formulary,6th edition,2004, Division of Pharmacology, UCT