The Relative Effectiveness of Three Full Kinetic Chain Treatment Protocols for Osteoarthritis of the Knee: Manual Therapy, Rehabilitation and a Combination thereof.

By

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I, Lauren Dwyer, do declare that this dissertation is representative of my own work in both conception and execution.

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DEDICATION

This dissertation is dedicated to my friends for all your love, encouragement and understanding throughout this project, and my studies. You have each, in your own way, helped me get to where I am today, with my sanity intact.

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ABSTRACT

Background:

Many treatment options provide symptomatic improvement of joint function for osteoarthritis (OA) of the knee. Research suggests full kinetic chain (FKC) manual and manipulative therapy (MMT) and rehabilitation yields greater benefits than home rehabilitation alone. However this treatment combination has never been compared against FKC MMT alone.

Objectives:

To determine the effectiveness of three FKC treatment protocols in the management of knee OA.

Method:

A single-blinded, randomised comparative trial of sixty-six patients with knee OA, equally allocated to three treatment groups: manipulation only, rehabilitation only or manipulation plus rehabilitation (a.k.a. combination group). Manipulation groups received bi-weekly FKC treatment, while a daily at-home stretching and exercise programme was prescribed to the groups receiving rehabilitation. Treatment lasted three weeks, with outcomes measure taken at baseline, pre-visit 4 and 1-week follow up. Primary outcome measures included the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC), and McMaster Overall Therapeutic Effectiveness (OTE) Tool.

Results:

There was a drop-out rate of 7.6% (n=5), with intent to treat analysis providing the missing data. All three treatment groups showed clinically and statistically significant changes in overall WOMAC scores from baseline to 1-week follow up. The combination group showed the largest improvement (50.5%), followed by manipulation (44.4%) and rehabilitation (33.6%). However, this difference between group improvement was not statistically significant (p= 0.156).

Conclusion:

All three intervention protocols showed statistically significant improvement in most outcome measures at 1-week follow-up. However, there was no statistically significant difference between groups and therefore it is concluded that the interventions appear to be equally effective in the short-term management of knee OA.

TABLE OF CONTENTS

DEDICATION ii
ACKNOWLEDGEMENTS iii
ABSTRACT iv
TABLE OF CONTENTS v
LIST OF APPENDICES x
LIST OF TABLES xi
LIST OF FIGURES xv
LIST OF DEFINITIONS xvi
LIST OF ABBREVIATIONS xviii
CHAPTER ONE 1
INTRODUCTION 1
1.1 The Problem and Its Setting 1
1.2 Aims of the Study 3
1.3 Objectives of the Study 4
1.4 Hypotheses of the Study 4
1.5 Purpose and Benefit of the Study 5
1.6 Limitations of the Study 6
1.7 Conclusion 6
CHAPTER TWO 7
LITERATURE REVIEW 7
2.1 Introduction 7
2.2 Anatomy 7
2.2.1 Bony Anatomy of the Knee Joint 7
2.2.2 Lower Limb Musculature 9
2.2.3 Lower Limb Biomechanics 11
2.2.3.1 Kinetic Chain Theory 12
2.3 Overview of Osteoarthritis of the Knee 13
2.3.1 Epidemiology 13
2.3.1.1 Incidence and Prevalence 13
2.3.1.2 Risk Factors 13
2.3.2 Pathology 16

2.3.2.1 Pathogenesis and Morphology 16
2.3.2.2 Natural History 18
2.3.2.3 Subsets of OA 18
2.3.3 Clinical Features 19
2.3.3.1 Signs and Symptoms 20
2.3.3.2 Arthrogenic Muscle Inhibition (AMI) 22
2.3.4 Diagnostic Criteria 23
2.3.4.1 Radiographic Diagnosis 24
2.3.4.2 Clinical Diagnosis 26
2.3.4.3 Differentiation from other Arthritic Diseases 26
2.4 Treatment Options 27
2.4.1 Pharmacologic Interventions 27
2.4.1.1 Medication 27
2.4.1.2 Surgery 29
2.4.2 Non-pharmacologic Interventions 29
2.4.2.1 Rehabilitation 29
2.4.2.2 Manual and Manipulative Therapy (MMT) 32
2.5 Conclusion 36
CHAPTER THREE 37
METHODOLOGY AND MATERIALS 37
3.1 Introduction 37
3.2 Study Design 37
3.3 Method 37
3.3.1 Advertising 37
3.3.2 Sample 38
3.3.2.1 Sample Size 38
3.3.2.2 Sample Allocation 38
3.3.3 Sample Characteristics 38
3.3.3.1 Inclusion Criteria 38
3.3.3.2 Exclusion Criteria 39
3.4 Clinical Procedure 41
3.4.1 Participant Assessment 41
3.4.1.1 Indications for the Application of FKC Manual Therapy 42
3.4.1.1.1 The Lumbopelvic Spine 42

3.4.1.1.2 The Hip Joint 42
3.4.1.1.3 The Knee Joint 42
3.4.1.1.4 The Ankle Joint 43
3.4.1.1.5 The Joints of the Foot 43
3.4.1.2 Radiographic Assessment 43
3.4.2 Interventions 44
3.4.2.1 Manipulative Therapy 44
3.4.2.2 Rehabilitation 44
3.5 Intervention Frequency 45
3.6 Measurement Tools 46
3.6.1 Subjective Data 46
3.6.1.1 Western Ontario and McMaster Universities Osteoarthritis Index 46
3.6.1.2 The McMaster Overall Therapy Effectiveness Tool 46
3.6.1.3 Beck Depression Inventory 46
3.6.1.4 Berg Balance Scale 47
3.6.2 Objective Data 47
3.6.2.1 Knee range of motion 47
3.6.2.2 One Legged Standing Test (OLST) 47
3.7 Measurement Frequency 48
3.8 Manual and Manipulative Therapy (MMT) Procedure 49
3.8.1 Lumbar Spine and Pelvis 49
3.8.2 Hip 49
3.8.3 Knee 49
3.8.4 Foot and Ankle 50
3.8.4.1 Distal Talofibular Joint 50
3.8.4.2 Tibiotalar Joint 51
3.8.4.3 General Midtarsal Joint 51
3.8.4.4 Tarsal Joint Plantar To Dorsal 51
3.8.4.5 First Metatarsophylangeal Joint and Sesamoid Bones 51
3.8.4.6 Second to Fifth Metatarsophylangeal and Intermetatarsal Joint 52
3.9 Rehabilitation Procedure 52
3.10 Statistical Methodology 54
CHAPTER FOUR 56
RESULTS 56

4.1 Introduction 56
4.2 The Data 57
4.2.1 Primary Data 57
4.2.2 Secondary Data 57
4.3 Physical Characteristics and Demographic Data 59
4.4 Baseline Readings 60
4.5 Assessment of the Treatment Effect 62
4.5.1 Intra-group Analysis 62
4.5.1.1 Subjective Outcomes Findings 62
4.5.1.1.1 Western Ontario and McMaster Universities Osteoarthritis Index 62
4.5.1.1.2 Beck Depression Inventory 67
4.5.1.1.3 Berg Balance Scale 67
4.5.1.1.4 McMaster Overall Therapy Effectiveness Tool 68
4.5.1.2 Objective Outcome Findings 70
4.5.1.2.1 One Leg Standing Test 70
4.5.1.2.2 Range of Motion 71
4.5.2 Inter-group Analysis 73
4.5.2.1 Subjective Data 73
4.5.2.1.1 WOMAC 73
4.5.2.1.2 Beck Depression Inventory 78
4.5.2.1.3 Berg Balance Scale 79
4.5.2.1.4 McMaster Overall Therapy Effectiveness Tool 80
4.5.2.2 Objective Data 82
4.5.2.2.1 One Leg Standing Test 82
4.5.2.2.2 Range of Motion 83
4.6 Summary 85
CHAPTER FIVE 87
DISCUSSION 87
5.1 Introduction 87
5.2 Demographics and Physical Characteristic 87
5.3 Comparison of Baseline Outcome Measurements 88
5.4 Assessment of Intervention Effect 89
5.4.1 Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) 89

5.4.2 Beck Depression Inventory (BDI)..... 93
5.4.3 Berg Balance Scale (BBS)..... 94
5.4.4 The McMaster Overall Therapy Effectiveness Tool (OTE) 95
5.4.5 One Leg Standing test (OLST) 95
5.4.6 Range of Motion 96
5.4.7 Summary of Findings..... 97
5.5 Study limitations..... 98
CHAPTER SIX 99
CONCLUSIONS AND RECOMMENDATIONS 99
6.1 CONCLUSIONS 99
6.2 RECOMMENDATIONS 99
LIST OF REFERENCES 101

LIST OF APPENDICES

Appendix A: Advertisement
Appendix B: Case History
Appendix C: Physical Examination
Appendix D: Lumbar Regional Examination
Appendix E: Hip Regional Examination
Appendix F: Knee Regional Examination
Appendix G: Foot and Ankle Regional Examination
Appendix H: S.O.A.P.E note
Appendix I: Letter of Information and Informed consent
Appendix J: Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC)
Appendix K: McMaster Overall Therapy Effectiveness (OTE) Tool
Appendix L: Beck Depression Inventory
Appendix M: Berg Balance Scale
Appendix N: Range of Motion
Appendix O: Medication and Exercise Diary
Appendix P: Exercise Brochure
Appendix Q: Randomised allocation chart
Appendix R: Ethics Clearance Certificate

LIST OF TABLES

CHAPTER 2

Table 2.1 Name Location and Functions of Knee Ligaments

Table 2.2.1 Attachments, Innervation and Action of Quadriceps Femoris Components

Table 2.2.2 Attachments, Innervation and Action of Hamstrings Components

Table 2.3 Movements of the Knee Joint

Table 2.4.1 Risk Factors and Protective Factors for Knee OA According To the Framingham OA Study

Table 2.4.2 Outline of Typical Symptoms of Osteoarthritis

Table 2.4.3 Outline of Clinical Signs Characteristic of Osteoarthritis

Table 2.4.4 Clinico-Radiographic Classification Criteria for Osteoarthritis of the Knee

Table 2.4.5 Clinico-Radiographic Classification Criteria for Osteoarthritis of the Hip

Table 2.4.6 Clinico-Radiographic Classification Criteria for Osteoarthritis of the Hands

Table 2.4.7 Kellgren-Lawrence Classification of Osteoarthritis

Table 2.4.8 Classification Criteria for Diagnosis of Idiopathic Osteoarthritis of the Knee

Table 2.4.9 Classification for Subsets of Idiopathic Osteoarthritis

Table 2.5 Medical Management of Symptomatic OA of Hip and Knee

CHAPTER 3

Table 3.1 Lists of Questions and Required Responses during Telephonic Interview

Table 3.2 Outline of the Treatment Protocols for Each Group

Table 3.3 Summary of Outcome Measure Requirements for Inclusion and Minimal Clinically Important Change (MCIC)

Table 3.4 Summary of Treatment and/or Measurement Frequency

Table 3.5 Knee Range of Motion Impairments of the Knee Addressed By Manual Therapy

Table 3.6 Strengthening Exercises for Rehabilitation Protocol

Table 3.7 Stretch Exercises for Rehabilitation Protocol

Table 3.8 Range Of Motion Exercises for Rehabilitation Exercises

CHAPTER 4

Table 4.1.1 Distribution of Sample According To Demographic Characteristics (Qualitative)

Table 4.1.2 Distribution of Sample According To Demographic Characteristics (Quantitative)

Table 4.2.1 Comparison of Baseline Outcomes between Groups
Table 4.2.2 ANOVA Comparison of Mean Baseline Outcomes between the Three Treatment Groups
Table 4.2.3 Bonferroni Post Hoc Comparison of Mean WOMAC Stiffness Scores Between the Three Treatment Groups
Table 4.3.1 Means and Standard Deviations in Overall WOMAC Scores at Baseline, Visit 4 and Visit 7
Table 4.3.2 Mean Difference and Percentage Change in Overall WOMAC Scores from Baseline to Visit 4 and Visit 7
Table 4.3.3 Intra-Group Comparison of the Effect on Overall WOMAC Scores of Each Intervention Group
Table 4.3.4 Means and Standard Deviations in WOMAC Pain Scores at Baseline, Visit 4 and Visit 7
Table 4.3.5 Mean Difference and Percentage Change in WOMAC Pain Scores from Baseline to Visit 4 and Visit 7
Table 4.3.6 Intra-Group Comparison of the Effect on WOMAC Pain Scores of Each Intervention Group
Table 4.3.7 Means and Standard Deviations in WOMAC Stiffness Scores at Baseline, Visit 4 and Visit 7
Table 4.3.8 Mean Difference and Percentage Change in WOMAC Stiffness Scores from Baseline to Visit 4 and Visit 7
Table 4.3.9 Intra-Group Comparison of the Effect on WOMAC Stiffness Scores of Each Intervention Group
Table 4.3.10 Means and Standard Deviations in WOMAC Disability Scores at Baseline, Visit 4 and Visit 7
Table 4.3.11 Mean Difference and Percentage Change in WOMAC Disability Scores from Baseline to Visit 4 and Visit 7
Table 4.3.12 Intra-Group Comparison of the Effect on WOMAC Disability Scores of Each Intervention Group
Table 4.3.13 Summary of Percentage Change in WOMAC Scores between Treatment Groups
Table 4.3.14 Means, Standard Deviations and Change in BDI Scores at Baseline, Visit 4 and Visit 7
Table 4.3.15 Intra-Group Comparison of the Effect on BDI Scores of Each Intervention Group
Table 4.3.16 Means, Standard Deviations and Change in BBS Scores at Baseline, Visit 4 and Visit 7

Table 4.3.17 Intra-Group Comparison of the Effect on BBS Scores of Each Intervention Group
Table 4.3.18 Means, Standard Deviations and Change in Overall Treatment Evaluation at Visit 4 and Visit 7
Table 4.3.19 Intra-Group Comparison of the Effect on OTE.1 Scores of Each Intervention Group
Table 4.3.20 Dichromatisation of Overall Treatment Evaluation at Visit 4 and Visit 7
Table 4.3.21 Means, Standard Deviations and Change in Overall Treatment Effect at Visit 4 and Visit 7
Table 4.3.22 Intra-Group Comparison of the Effect on OTE.2 Scores of Each Intervention Group
Table 4.4.1 Means, Standard Deviations and Change in OLST Scores at Baseline, Visit 4 and Visit 7
Table 4.4.2 Intra-Group Comparison of the Effect on OLST Scores of Each Intervention Group
Table 4.4.3 Means, Standard Deviations and Change in ROMf Readings at Baseline, Visit 4 and Visit 7
Table 4.4.4 Intra-Group Comparison of the Effect on ROMf Scores of Each Intervention Group
Table 4.4.5 Intra-Group Comparison of the Effect on ROMe Scores of Each Intervention Group
Table 4.4.6 Intra-Group Comparison of the Effect on ROMe Scores of Each Intervention Group
Table 4.4.7 Summary of Unit Change in Outcome Scores between Treatment Groups
Table 4.4.8 Summary of Minimum Clinically Indicated Change for Each of the Outcomes Using In This Study
Table 4.5.1 Inter-Group Comparison of Time Effects for Overall WOMAC
Table 4.5.2 Inter-Group Comparison of Time Effects for WOMAC Pain Score
Table 4.5.3 Inter-Group Comparison of Time Effects for WOMAC Stiffness Score
Table 4.5.4 Inter-Group Comparison of Time Effects for WOMAC Disability Score
Table 4.5.5 Inter-Group Comparison of Time Effects for BDI
Table 4.5.6 Inter-Group Comparison of Time Effects for BBS
Table 4.5.7 Inter-Group Comparison of Time Effects for OTE.1
Table 4.5.8 Kruskal-Wallis Test to Compare Median OTE.2 Scores Between the Three Treatment Groups
Table 4.6.1 Inter-Group Comparison of Time Effects for OLST
Table 4.6.2 Inter-Group Comparison of Time Effects for ROMf

Table 4.6.3 Inter-Group Comparison of Time Effects for ROMe

Table 4.7.1 Summary of the Level of Statistical Significance, Clinical Significance and Overall Change of Outcomes Measures in Each Intervention Group from Baseline to Final Follow-Up.

CHAPTER 5

Table 5.1 Distribution of Sample According To Demographic Characteristics (Quantitative)

Table 5.2 Summary of WOMAC Outcomes Results

Table 5.3 Summary of Subjective and Objective Outcomes

LIST OF FIGURES

CHAPTER 2

Figure 2.1 Anterior View of the Bony Anatomy of the Knee Joint with Ligaments and Menisci
Figure 2.2 Posterior View of the Bony Anatomy of the Knee Joint with Ligaments and Menisci
Figure 2.3 Anterior View of the Muscles of the Thigh
Figure 2.4 Posterior View of the Muscles of the Thigh

CHAPTER 4

Figure 4.1 Modified Consolidated Standard for Reporting Clinical Trials (Consort) Diagram
Figure 4.2 Mean Overall WOMAC Score over Time by Treatment Group
Figure 4.3 Mean WOMAC Pain Score over Time by Treatment Group
Figure 4.4 Mean WOMAC Stiffness Score over Time by Treatment Group
Figure 4.5 Mean WOMAC Disability Score over Time by Treatment Group
Figure 4.6 Mean BDI Score over Time by Treatment Group
Figure 4.7 Mean BBS Score over Time by Treatment Group
Figure 4.8 Mean OTE.1 Score over Time by Treatment Group
Figure 4.9 Boxplot of OTE.2 Scores by Group and Time Point
Figure 4.10 Mean OLST Score over Time by Treatment Group
Figure 4.11 Mean ROMf Score over Time by Treatment Group
Figure 4.12 Mean ROMe Score over Time by Treatment Group

LIST OF DEFINITIONS

Chiropractic Adjustment

Described as a specific form of articular manipulation characterised by direct (short lever), specific, high-velocity, low amplitude thrust, usually associated with restored range of motion, pain-free mobility and improved function following its application (Bergmann and Peterson, 2002).

Clinical Prediction Rules (CPRs)

Clinical prediction rule are a clinical tool developed to identify what influence a combination of certain medical sign, symptoms, and other clinical outcomes have on the diagnosis, prognosis or probability of a specific disease or result (Herbert and Fritz, 2012).

Degenerative Joint Disease (DJD)

Degenerative joint disease and osteoarthritis are used interchangeably, and are terms which refer to a progressive cartilage loss, with the resultant development of bony spurs and cysts at the joint margins (Kumar *et al.,* 2007).

Gate Control Theory

Melzack and Wall (1965) proposed that, due to the composition of nerve fibres carring information from the site of injury to the spinal cord, less pain is felt when more large fibre (touch, pressure, vibration) activity relative to thin fibre (pain) activity is received at the inhibitory cell.

Joint dysfunction

A joint dysfunction is a disturbance of function without structural change, thereby affecting quality and range of motion. It can present as a decrease in motion, increase in motion or an abnormal motion (Bergmann and Peterson, 2002).

Joint receptors

Joint receptors are specialised cells or sub-cellular structures that change their properties in response to various stimuli (Hopkins and Ingersoll, 2000).

Kinetic Chain

Defined as “a combination of several successively arranged myofascial, articular and neural components, constituting a complex unit” (Bergmann and Peterson, 2002;

Sahrmann, 2010). The full kinetic chain of the lower limb included all joints from the sacroiliac to those in the foot.

Manual and Manipulative Therapy

The therapeutic application of manual force, including all procedures in which a manual therapist uses the hands to mobilise, adjust, manipulate, apply traction, massage, stimulate, or otherwise directly or indirectly influence the patient's function and/or health (Gatterman, 2005).

Mechanoreceptor

A receptor that is excited by mechanical pressures or distortions such as sound, touch and movement (Redwood, 1997).

Myofascial Trigger Points (MTPs)

Myofascial trigger points are defined as a hyper-irritable location within a taut band of skeletal muscle that is painful when compressed and can give rise to characteristic referred pain, tenderness and tightness. Cumulatively, MTPs result in myofascial pain syndrome (Chaitow and DeLany, 2005).

Nociceptor

Pain receptors which detect damage occurring within tissues due to physical, mechanical or chemical damage (Redwood, 1997).

Substance-P

Information about tissue damage is received by nociceptors and transmitted by substance-P, a neuropeptide, to the central nervous system, where it is converted to pain sensation (Melzack and Wall, 1965).

Type I Muscle Fibre

Type I, or slow twitch, muscle fibres are better suited to utilise oxygen for energy generation, and therefore have long periods of sustained contraction, but with little force (Caroll *et al.,* 2006).

Type II Muscle Fibre

Type II, or fast twitch, muscle fibres contract rapidly and with greater force, but fatigue quickly due to their anaerobic activity and lactic acid build up (Caroll *et al.,* 2006).

LIST OF ABBREVIATIONS

ANOVA	Analysis of Variance
BBS	Berg Balance Scale
BDI	Beck Depression Inventory
BMI	Body Mass Index
cm	Centimetre
CPPD	Calcium Pyrophosphate Dihydrate
CPR	Clinical Predictive Rules
DIP/J	Distal Interphylangeal / Joint
DJD	Degenerative Joint Disease
DMOAD/s	Disease Modifying Anti-Osteoarthritis Drug/s
EMG	Elecromyograph
ESR	Erythrocyte Sedimentation Rate
FKC	Full Kinetic Chain
HVLA	High Velocity Low Amplitude
kg	Kilogram
MCIC	Minimum Clinically Indicated Change
mm	millimetre
MMT	Manual and Manipulative Therapy
mo	Months
n	Sample Size
NSAID/s	Non-steroidal Anti-inflammatory Drug/s
OA	Osteoarthritis
OLST	One Leg Standing Test
OTE	McMaster Overall Therapeutic Effectiveness Tool
p	Level of Significance
PFPS	Patellofemoral Pain Syndrome
PIP/J	Proximal Interphylangeal / Joint
RA	Rheumatoid Arthritis
ROM	Range of Motion
SD	Standard Deviation
SMT	Spinal Manipulative Therapy
VAS	Visual Analogue Scale
WOMAC	Western Ontario and McMaster Universities Osteoarthritis Index
Yrs	Years

CHAPTER ONE

INTRODUCTION

1.1 The Problem and Its Setting

Osteoarthritis (OA) is a chronic degenerative joint disorder with a complex aetiology (Felson, 2000). It is characterised by focal loss of articular cartilage within synovial joints, associated with hypertrophy of bone and thickening of the capsule, resulting in alterations in biomechanical properties of the joint (Woolf and Pfleger, 2003). This common disorder affects mostly people above the age of 60, occurs in any joint but most commonly in the hip, knee, hand, foot, and spine (Symmons, Mathers and Pfleger, 2003); with as many as 40% of people over the age of 65 suffering symptoms associated with knee or hip OA (Zhang *et al.*, 2008). Although no cure exists, a number of surgical, pharmacological and non-pharmacological treatment options may provide symptomatic relief and improvement of joint function (Sarzi-Puttini *et al.*, 2005). Amongst the non-pharmacological interventions, rehabilitation and manual therapies appear to be the most beneficial in promoting knee function (Deyle *et al.*, 2005).

McCarthy *et al.* (2004) compared the effectiveness of an at-home exercise programme alone or supplemented with a class-based exercise programme in patients with knee OA. A greater improvement in Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) score was found in the class-based exercise group (20.6%) versus the at-home group (8.8%). WOMAC scores were used to detect changes in the patients' perception of their ability to function and their quality of life, specifically related to the disease process. These relatively modest effects on WOMAC scores may be related to the inability of exercise to address a number of factors that prevent patients from maximising the benefits from their exercise programme (Fitzgerald, 2005). Possible examples of such contributing factors were identified as: 1) quadriceps inhibition or activation failure, 2) obesity, 3) passive knee laxity, 4) knee misalignment, 5) fear of physical activity and 6) self-efficacy (Fitzgerald, 2005).

In a more current investigation, Jan *et al.* (2009) investigated the effectiveness of weight-bearing versus non-weight-bearing exercise on function, walking speed, and proprioception (joint position sense) in participants with knee OA. Their study showed that simple knee flexion and extension exercises (weight-bearing and non-weight-bearing) significantly improved knee strength and functional capacity (viz. WOMAC scores).

Weight-bearing exercises further increased proprioception, which commonly decreases with age and predisposes individuals to disturbed postural control and falls (Sturnieks, St. George. and Lord, 2008). Therefore, based on these results, it may be possible that applying exercise intervention to the full kinetic chain (FKC) will increase lower limb strength and proprioception, resulting in increased balance and decreased risk of a fall (Sturnieks, St. George, and Lord, 2008; Jan *et al.*, 2009).

In an earlier study, Tucker, Brantingham and Myberg (2003) compared the effectiveness of knee joint manipulation versus non-steroidal anti-inflammatory drugs (NSAIDs), and found manipulation to be just as effective as NSAIDs in the treatment on knee OA. NSAIDs have been shown to be more effective than a placebo in relieving knee OA pain (Bjordal *et al.*, 2004); but, NSAIDs have many adverse side-effects (Wolfe, Lichtenstein, and Singh, 1999). Fish *et al.* (2008) had similar results when comparing the effectiveness of knee joint mobilisation against topical capsaicin cream. Capsaicin cream had previously been demonstrated as a superior treatment option to a placebo in painful disorders (viz. knee OA and general osteoarthritis) (Gemmell, Jacobson, and Hayes, 2003). Based on these results, it seems that both manipulation and mobilisation of the knee joint are more effective than a placebo in the treatment of knee OA.

Pollard *et al.* (2008) applied a manipulative therapy protocol to symptomatic knee joint complexes (patellofemoral articulation), finding a statistically significant improvement in knee pain, mobility, crepitus and function as compared to the placebo-control group[1]. It was noted that the knee treatment also had a significant improvement in hip movement of those in the intervention group compared to the placebo-control group. This was attributed to effects that single joint treatment has on the FKC (Pollard *et al.*, 2008; Brantingham *et al.,* 2009). Studies supporting the FKC theory include findings where treatment was applied to various joints of the lower extremity kinetic chain to determine their effect on the knee. These studies suggest that correcting biomechanical abnormalities in hip (Cliborne *et al.* 2004; Currier *et al.*, 2007) and lumbopelvic (Iverson *et al.*, 2008) regions yield favourable effects on knee joint pain / dysfunction. There is, however, a paucity of literature to determine whether these favourable changes would be evident in knees that present with a degenerative pathology (viz. knee OA).

Additionally, few studies have looked at what effect combining manipulation and rehabilitation would have in the treatment of knee OA. Deyle *et al.,* (2000) applied manual

[1]Placebo was defined as interferential current set at zero.

and manipulative therapy (MMT) to the knee as well as to the lumbar spine, hip and ankle as required. Patients were also given a knee exercise programme to perform in the clinic on treatment days and daily at-home. WOMAC scores improved by 55.8% in the treatment group as compared to a 14.6% improvement in those patients receiving placebo treatment[2], thus showing the effectiveness of combining manipulation and rehabilitation. Five years later, using similar methodologies, Deyle *et al.*, (2005) compared an at-home versus in-clinic physical therapy programme. Participants treated in-clinic received supervised exercise, MMT to the FKC and a home exercise programme, while a second group received at-home exercise only. Significant improvements were seen in both groups; however, the in-clinic treatment group had twice the improvement in WOMAC scores as compared to the at-home exercise group. The researcher attributed this difference between groups to the application of MMT to the full kinetic chain. However, the clinic group performed the exercises under supervision and were corrected when necessary while the home group were largely unsupervised and may have performed the exercises incorrectly, thus decreasing the benefit such exercises would have had. One cannot, therefore, consider that the difference in group performance would be solely attributed to the addition of MMT.

To date there does not seem to have been a study which compares the effect of FKC MMT alone versus the FKC MMT plus rehabilitation or FKC rehabilitation alone. Therefore a need existed for a study to determine whether FKC MMT combined with a standardised rehabilitation programme is more effective than either intervention alone in the treatment of knee OA.

1.2 Aims of the Study

The aim of this investigation was to determine the relative effectiveness of FKC MMT, FKC rehabilitation, and a combination of the FKC MMT and FKC rehabilitation, in terms of subjective and objective clinical findings, for the management of OA of the knee.

[2]Placebo was defined as subtherapeutic ultrasound.

1.3 Objectives of the Study

The objectives of this study were:

1) To determine the relative effectiveness of:
    a) Manipulation
    b) Rehabilitation
    c) Manipulation and rehabilitation

    In terms of subjective findings [the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC), patient rated satisfaction in terms of the McMaster Overall Therapy Effectiveness (OTE) Tool, the Beck Depression Inventory (BDI) and the Berg Balance Scale (BBS)] in the short-term management of knee OA

2) To determine the relative effectiveness of:
    a) Manipulation
    b) Rehabilitation
    c) Manipulation and rehabilitation

    In terms of objective findings [One Leg Standing Test (OLST) and inclinometer measures (ROMf and ROMe)] in the short term management of knee OA

3) To compare short-term results between the three treatment groups in order to determine which treatment is most effective in terms of subjective and objective measurements.

1.4 Hypotheses of the Study

Null Hypothesis One:

It was hypothesised that there would be no significant difference between treatment groups in terms of in short-term subjective findings.

Null Hypothesis Two:

It was hypothesised that there would be no significant difference between treatment groups in terms of in short-term objective findings.

Null Hypothesis Three:

It was hypothesised that no treatment group was more effective in the short-term management of knee OA.

1.5 Purpose and Benefit of the Study

OA is a common worldwide condition, the prevalence of which affects 9.6% of men and 18% of women over the age of 60 years (Woolf and Pfleger, 2003). However, despite the extensive pharmacological and non-pharmacological treatments available, to date there is no one treatment that has been clinically demonstrated to be superior.

Application of MMT and rehabilitation treatment to the FKC was found to be more effective than a placebo (Deyle *et al.*, 2000) and home rehabilitation alone (Deyle *et al.*, 2005) in reducing joint pain, stiffness and dysfunction associated with OA of the knee. However, this treatment combination has not (according to literature), been compared against FKC MMT alone. If FKC MMT alone is found to be effective treatment for knee OA, this knowledge would allow Chiropractors to manage patients without having to rely on patient compliance to a rehabilitation protocol.

In combination, the therapeutic action of MMT and rehabilitation is likely to have an effect on both the static (bone, cartilage and ligaments) and dynamic (muscles and tendons) structures of the joint. Thereby having an influence on all involved structures in a joint pathology. However, there is a paucity of knowledge regarding the effect of MMT on surrounding musculature, and similarly rehabilitation on the static structures (Deyle *et al.*, 2005).

An additional consideration is that the elderly are prone to falls, which, while multi-factorial, cause nearly two-thirds of all non-intentional injury related deaths in older adults (Hawk *et al.,* 2006). The major intrinsic risk factors for instability include lower extremity dysfunction resulting in altered proprioception, and disorders of gait and balance (Gleberzon, 2001). Joint degeneration, causing structural changes to joint mechanoreceptors, thus results in loss of hip and knee proprioception, leading to an increased chance of falls (Bowers, 1996). Education and therapy have found a 7-12% reduction in risk of a fall by targeting potential risk factors (Gleberzon, 2001), which is especially important in light of the fact that proximal hip fractures resulting from falls have been found to be associated with an aging population (Parker and Pryor, 1993). Thus

treatments like rehabilitation and/or MMT may improve proprioception, leading to a decreased risk of falls experienced by the elderly.

1.6 Limitations of the Study

As with all studies that utilise subjective outcomes as part of the measurement and reporting process, it is assumed that all patients answered the questions openly and honestly, and therefore, reflected their condition accurately at the time of measurement (Mouton and Babbie, 2006).

Whilst every effort was made at the initial consultation to educate the patient about their prescribed at-home rehabilitation protocol, it is possible that the exercises were not properly carried out or that they did not fully comply with the exercise prescription.

1.7 Conclusion

This research aimed to contribute to the body of knowledge pertaining to the kinetic chain of the lower limb and its relationship to osteoarthritis of the knee. This research may further contribute to the growing body of literature related to the use of the kinetic chain when assessing and treating chronic joint dysfunction and degeneration.

In the remaining chapters, the researcher will review the pertinent literature related to the topic (Chapter Two); describe in detail the methodology of this study (Chapter Three) and present the statistical results (Chapter Four). The subsequent conclusions (Chapter Five) and suggested recommendations will conclude this study.

CHAPTER TWO

LITERATURE REVIEW

2.1 Introduction

The following chapter intends to provide an overview of current literature pertinent to this study. A brief outline of related anatomy and biomechanics of the knee joint will be discussed, as well as a theoretical association of knee osteoarthritis and the lower kinetic chain. The clinical, aetiological and epidemiological aspects of osteoarthritis of the knee will be provided, along with the potential effects that various treatment options may have on this disease.

2.2 Anatomy

2.2.1 Bony Anatomy of the Knee Joint

The knee joint functions primarily as a large hinge-type joint, consisting of three articulations, two tibiofemoral articulations between the medial and lateral femoral and tibial condyles, and one patellofemoral articulation between the posterior aspect of the patella and femur (Moore and Dalley, 2006). Due to the relative incongruence of the articulating surfaces, the knee joint complex is inherently unstable, therefore two fibrocartilage discs (menisci) exist in the space between the tibia and femur, which are attached to the intercondylar eminence of the tibia, and further increase the congruency of the joint as well as provide additional stability (Magee, 2008).

In addition to the menisci, numerous surrounding ligaments play an important role in stabilisation of the knee. The name, location and specific function of these ligaments are outlined in Table 2.1.

Table 2.1 Name, Location and Functions of Knee Ligaments

Name	Attachments	Function
Medial collateral ligament (MCL)	Descends from its attachment on the medial femoral epicondyle and the adductor tubercle to the medial margin and medial surface of the tibia, superior and posterior to the attachment of sartorius, gracilis, and semitendinosus tendons. Its deep fibres attach to the fibrous membrane of the underlying medial meniscus.	Prevents abduction of the tibia on the femur, and stabilises the hinge-like motion of the knee joint.

Table 2.1 continued. Name, Location and Functions of Knee Ligaments

Lateral collateral ligament (LCL)	Attaches to the lateral femoral epicondyle, superior to the groove for the popliteus tendon, and descends to the lateral surface of the fibular head. It is separated by a bursa from the fibrous membrane of the lateral meniscus.	It is stronger than the MCL in its ability to prevent adduction of the tibia on the femur, and stabilises the hinge-like motion of the knee joint.
Anterior cruciate ligament (ACL)	Ascends posteriorly from the anterior facet of the tibial spine to the facet at the back of the lateral wall of the intercondylar fossa of the femur	Prevents hyper-extension and excessive internal rotation, when knee in extension. In flexion, prevents the anterior translation of the tibia on the femur.
Posterios cruciate ligament (PCL)	Descends from the anterior aspect of the intercondylar notch of the femur, attaching posteriorly on the tibial plateau, distal to the joint line	In extension, prevents the posterior translation of the tibia on the femur.
Articular capsule and the bursae	The joint capsule surrounds only the posterior, medial and lateral aspects of the femoral and tibial condyles. The capsule consists of an external fibrous capsule and an internal synovial membrane, which is continuous with the synovial lining of the bursae of the knee.	The bursae act to reduce friction between the tendons and underlying bones. The articular capsule is stabilised and strengthened by the joint ligaments and the muscle tendons of the knee.

(Table adapted from Moore and Dalley, 2006; Drake, Vogl and Mitchell, 2008; and Standring, 2008)

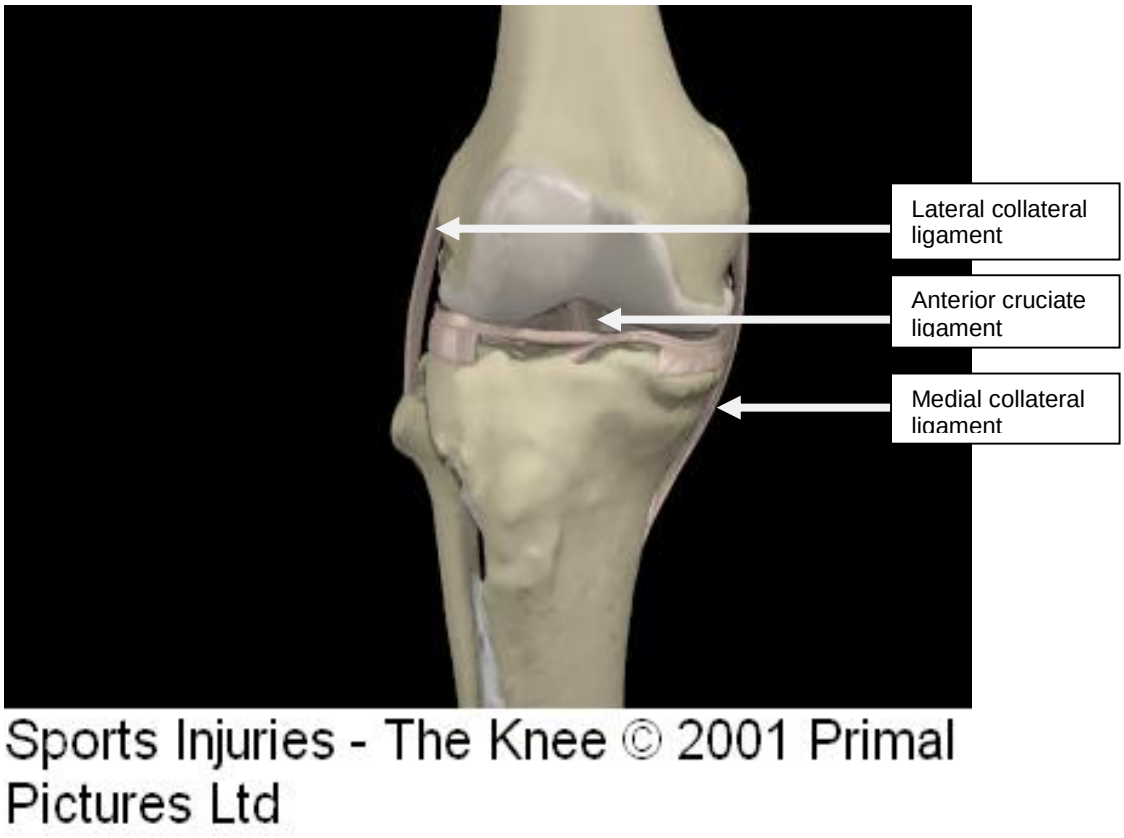

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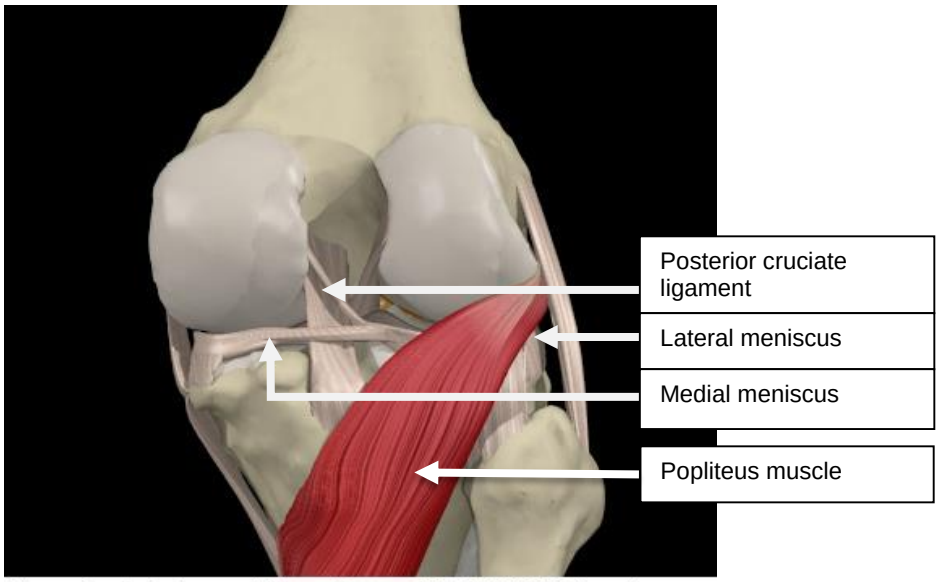
Figure 2.1 Anterior View of the Bony Anatomy of the Knee Joint with Ligaments and Menisci



Sports Injuries - The Knee © 2001 Primal Pictures Ltd

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Figure 2.2 Posterior View of the Bony Anatomy of the Knee Joint with Ligaments and Menisci

2.2.2 Lower Limb Musculature

The primary muscle groups responsible for creating movement at the knee joint are the quadriceps femoris, responsible for extension, and the hamstring muscle group, responsible for flexion. The attachments, innervation and action of the individual muscles within the quadriceps femoris and hamstring muscle groups are described in Table 2.2.1 and Table 2.2.2, respectively.

Table 2.2.1 Attachments, Innervation and Action of Quadriceps Femoris Components

Muscle	Proximal Attachment	Distal Attachment	Innervation	Action
Rectus Femoris	Anterior inferior iliac spine and ilium superior to the acetabulum	Via a common tendinous insertion to the base of the patella; indirectly via the patellar ligament to the tibial tuberosity	Femoral Nerve (L2, L3, L4)	Extend leg at knee joint; rectus femoris also steadies hip joint and helps iliopsoas muscle flex the thigh
Vastus Lateralis	Greater trochanter and lateral lip of linea aspera of the femur			
Vastus Medialis	Inter-trochanteric line and medial lip of linea aspera of the femur			
Vastus Intermedius	Anterior and lateral surfaces of shaft of the femur			

(Table adapted from Moore and Dalley, 2006; Drake, Vogl and Mitchell, 2008; and Standring, 2008)

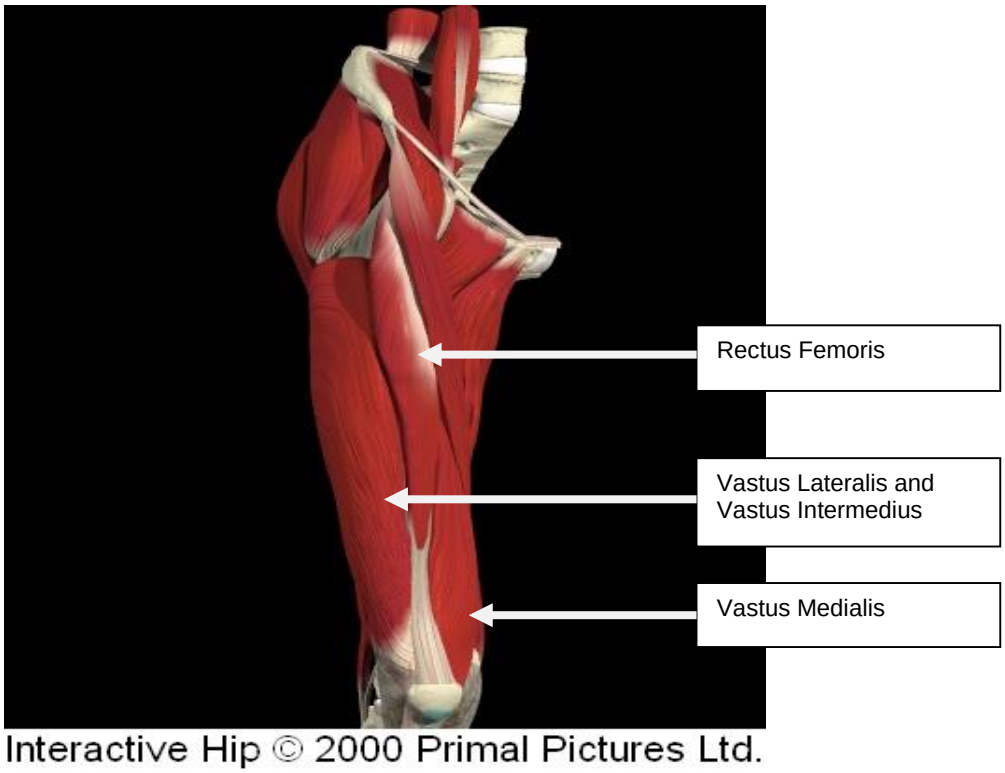

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Figure 2.3 Anterior View of the Muscles of the Thigh

Table 2.2.2 Attachments, Innervation and Action of the Components Related to the Hamstrings and Posterior Thigh

Muscle	Proximal Attachment	Distal Attachment	Innervation	Action
Semi-tendinosus	Ischial tuberosity	Superior part of tibia on medial surface	Tibial division of sciatic nerve (L5, S1, S2)	Extend thigh; flex leg and rotate it medially when knee is flexed
Semi-membranosus	Ischial tuberosity	Posterior part of tibia on medial condyle		
Biceps Femoris	Long head: ischial tuberosity Short head: linea aspera and lateral supracondylar line of femur	Fibula on lateral side of head	Long head: Tibial division of sciatic nerve (L5, S1, S2) Short head: Common fibular division of sciatic nerve (L5, S1, S2)	Extend thigh; flex leg and rotate it laterally when knee is flexed
Popliteus	Lateral aspect of lateral epicondyle of femur, outer margin of the lateral meniscus (intracapsular)	Posterior part of tibia, above soleal line	Tibial nerve (L4, L5, S1)	Assists flexing the leg; when leg flexed, it medially rotates the tibia

(Table adapted from Moore and Dalley, 2006; Drake, Vogl and Mitchell, 2008; and Standring, 2008)

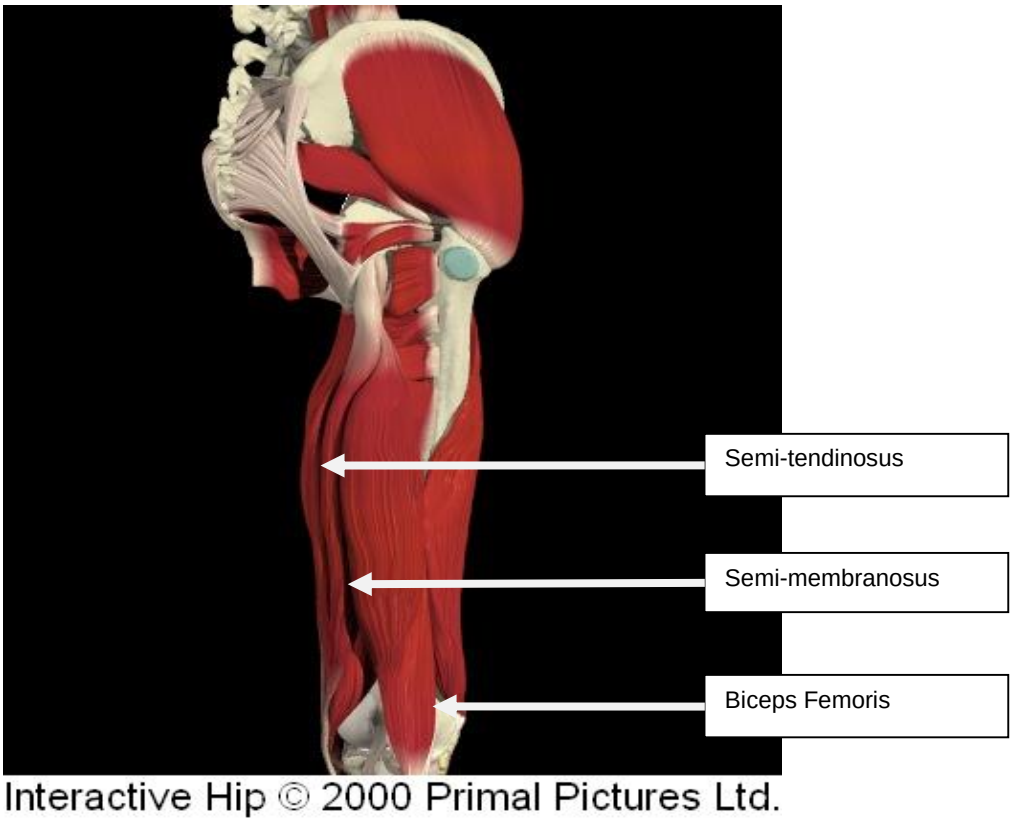

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Figure 2.4 Posterior View of the Muscles of the Thigh

2.2.3 Lower Limb Biomechanics

The primary movements of the knee are flexion and extension, with a smaller rotational component when the knee is flexed (Moore and Dalley, 2006). Table 2.1.3 outlines the main movements of the knee joint, as well as the name and action of the muscle producing these movements.

Table 2.3 Movements of the Knee Joint

Movement and range of movement	Name	Action
Knee flexors 135°-150°	1. Hamstrings (consisting of the semi-tendinosus, semi-membranosus, and biceps femoris) 2. Popliteus	Flexes and medially rotates the knee, locks and unlocks the knee from the flexed position.
Knee extensors 0°-10°	Quadriceps femoris (consisting of the rectus femoris, vastus lateralis, vastus medialis, vastus intermedialis)	Extends knee, (but flexes hip by action of rectus femoris).
Medial rotation 0°-10°	Popliteus (non-weight bearing when knee extended), or semi-tendinosus and semi-membranosus (when knee is flexed)	Unlocks knee by rotating femur 5° laterally on fixed tibia. Assists with knee flexion.
Lateral rotation 0°-30°	Biceps femoris (when knee is flexed)	Unlocks knee by rotating femur 5° medially on fixed tibia

(Adapted from Reid, 1992; Evans, 2001; Levangie and Norkin, 2001)

The relative incongruence of the articular surface results in the knee being relatively weak mechanically, and a greater reliance is placed on the actions of surrounding muscles, tendons and ligaments for strength and support (Magee, 2008). The anterior thigh muscles are the most vital of these supports, with the quadriceps femoris muscles being the most important stabiliser of the knee joint (Evans, 2001; Moore and Dalley, 2006).

A difference in activation patterns in the vastus medialis and biceps femoris muscles have been recorded during stair-walking and, to a lesser extent, level-walking in patients with knee OA. A possible reason for the altered activation patterns occurring may result from disease specific compensatory mechanisms, which become more apparent with more difficult tasks (Liikavainio *et al*., 2010).

A study by Astephen Wilson *et al*. (2011) aimed to investigate whether there was a correlation between radiographic change or pain severity and knee joint biomechanics and neuromuscular control in individuals clinically diagnosed with moderate knee OA. It was found that higher pain scores were associated with lower gait speeds and altered neuromuscular patterns.

Therefore, it is evident that patients with knee OA are likely to compensate in their gait patterns by utilising different combinations of joint motion (Evans, 2001) which has led to the development of the Kinetic Chain Theory.

2.2.3.1 Kinetic Chain Theory

The kinetic chain is defined as “a combination of several successively arranged myofascial, articular and neural components, constituting a complex unit” (Bergmann and Peterson, 2002; Sahrmann, 2010). There are 3 sub-systems within a kinetic chain (active, passive and neural), all of which contribute to the production of movement (Panjabi and White, 2001; Sahrmann, 2010). The passive sub-system comprises the bones, cartilage and ligaments of the lower limb, which provide sites for attachment of muscles and tendons (active sub-system), as well as provide protection to some of the structures making up the neural sub-system (Panjabi, 1992). This movement system relies not only on the alignment and mechanics of each joint in the chain, but also the co-ordinated recruitment muscles surrounding the joints to create active stabilisation and movement patterns (Panjabi and White, 2001; Sahrmann, 2010).

In the lower limb, a functional relationship exists between the knee and the superior articulations in the hip and lumbopelvic spine, as well as inferiorly to the foot and ankle (Leach, 2003). When the feet are weight-bearing, the kinetic chain is closed and the links function interdependently, with a change in one joint resulting in an immediate effect on the kinetics of other joints in the chain. Therefore, a dysfunction in the knee can have a direct effect on adjacent joints in the chain, and vice versa (Forbes, 2009).

This interdependence highlights the importance of addressing not only the area of complaint, but also adjacent regions to correct any potentially altered biomechanics of the kinetic chain in totality.

2.3 Overview of Osteoarthritis of the Knee

2.3.1 Epidemiology

2.3.1.1 Incidence and Prevalence

Knee cartilage will show histological changes in nearly 100% of the population over the age of 60. Of this population group, at least one joint will display radiographic evidence of OA in over 80%, approximately 40% will present with clinical symptoms of OA, and activity limitation will be present in 10% (Loeser, 2000).

2.3.1.2 Risk Factors

According to the Framingham Osteoarthritis Study (Felson, 1990), the major risk factors for knee OA are age, female gender, obesity, non-smoking, occupational knee bending, physical labour, and chondrocalcinosis. Other risk factors identified are listed in Table 2.4.1.

Table 2.4.1 Risk Factors and Protective Factors for Knee OA According to the Framingham Osteoarthritis Study

Risk factors	
Age	Immobilisation
Congenital/developmental defects	Joint hypermobility and instability
Crystal deposition in joints	Major joint trauma
Ethnic Group	Obesity
Female gender	Occupational knee bending
Genetics	Peripheral neuropathy
Geographic	Prior inflammatory joint disease
High bone mineral density	Repetitive joint use
Contradictory evidence	
Diabetes	
Oestrogen excess	
Hypertension	
Hyperuricaemia	
Protective factors	
*Smoking	
Osteoporosis	
Weight reduction	

(Adapted from Felson, 1990; Nevitt and Felson, 1996; Taylor and Resnick, 2000)

* Later studies have provided inconclusive evidence as to the effect of smoking on OA; with some considering it to decrease the risk of total joint replacement (Mnatzaganian *et al*., 2011) and others found available data to be inconclusive (Blagojevicy *et* al., 2010 and Hui *et al*., 2011).

In order to contextualise these factors for this study, some of the above factors will be discussed, particularly as they relate to some of the methodological considerations outlined in Chapter Three, and therefore, an understanding of these factors in context is required.

Age: Age is the strongest risk factor for OA, with an increase in prevalence of symptomatic OA from 7.0% in people aged 63-69 years old to 11.2% in people over the age of 80. Radiographic evidence of OA increased from 27.4% amongst people in their sixties compared to 43.7% prevalence in people in their eighties (Felson, 1990; Cooper *et al.,* 2000).

Gender: Age related increases in OA are found to be more apparent in females; not only with regard to incidence, but also in severity and rate of progression; moderate to severe OA changes increased in prevalence by 7.9% from the sixth to eighth decade of life (Felson, 1990). Although there was little or no difference in gender prevalence of mild OA (Roberts and Burch, 1996), females tended to have more severe OA, a greater number of joints involved, more symptoms, and a higher prevalence of hand and knee OA (Kellgren, Lawrence and Bier, 1963). Recent studies suggest that post-menopausal oestrogen deficiency may play a role in development of knee OA in older women (Nevitt and Felson, 1996). Males, however, had an increased prevalence of hip OA, especially in people aged 55 and above (Kellgren, Lawrence and Bier, 1963).

Obesity: Obesity is the strongest modifiable risk factor for development of knee OA, especially in women (Loeser, 2000). According to the Framingham OA Study, higher body mass index (BMI) was associated with a 61.5% increased risk of developing OA per 5-unit increase in BMI. Similarly, a weight loss correlated to a 40% decrease in risk of knee OA per 4.5 kg weight loss (Felson *et al*., 1997). Risk for development of knee OA increased exponentially when obesity was present with an additional risk factor, such as heavy physical activity. Elderly patients in the upper tertile of BMI, who performed at least three hours of daily physical activity, had a 92.9% risk of developing knee OA (McAlindon *et al*., 1999).

Major joint trauma: The relative risk for development of radiographic knee OA following meniscectomy for management of isolated meniscal tears was 93.3% (Roos *et al*., 1998). Studies also suggest that quadriceps weakness increased the risk of both radiographic and symptomatic OA (Slemenda *et al*., 1997).

Repetitive joint use: While there is insufficient data to suggest that sporting activities may lead to generalised OA (Lane and Buckwalter, 1993), it has been shown that certain occupations may cause the overuse of particular joints, thus increasing the risk of development of localised OA (Croft *et al*., 1992). For example, occupational knee bending is strongly associated with knee OA and miners often exhibit signs of spondylosis (Felson, 1990).

Muscle Imbalance and Atrophy: Muscles play a major role in joint biomechanics as they produce movements, absorb loading, and provide dynamic joint stability. It is, therefore, possible that muscle weakness due to aging or prior trauma may result in loss of protective muscle control, excessive joint movement and instability (Slemenda *et al*., 1997; Armin *et al.*, 2009). Ultimately, this will cause stress-induced microtrauma of the articular cartilage due to the increased occurrence in physiological shear and peak joint forces. Over an extended period of time, this microtrauma will cause cartilage degeneration, with pathological subchondral pressure increase and consequent subchondral sclerosis, and joint collapse with axis mal-deviation (Ingersoll, Palmieri and Hopkins, 2003).

Slemenda *et al.* (1998) conducted a prospective study in which reduced knee extensor strength was present in subjects who developed OA as compared to the unaffected participants. Similar findings were seen in a study on patients with unilateral ankle OA, in

which the affected side displayed reduced calf circumference and decreased electromyography (EMG) frequencies of lower leg muscles (Valderrabano *et al*., 2006).

In a healthy individual, muscle biopsies have shown atrophy of type-I muscle fibres (slow-twitch) in the vastus lateralis following periods of knee immobilisation. In knee OA patients however, weakness of the vastus lateralis was mostly due to type-II fibre (fast-twitch) atrophy (Nakamara and Suzuki, 1992). Fink *et al*. (2007) then investigated the structural changes in the vastus medialis and found type-II fibre atrophy in all subjects as well as additional type-I fibre atrophy in 32% of the patients. Exercise training has been found to increase the diameter of both type-I and type-II muscles fibres (Saltin *et al*., 1977), and therefore these researchers recommended exercise as a counter-action against muscle atrophy and thus delay the onset of OA.

As muscles increase in size with exercise, it is suspected that atrophy in arthritic patients is not only caused by disuse in the presence of joint stiffness and pain, but also by age-related sarcopenia (generalised loss of skeletal muscle mass), physical immobilisation and reduced physical activity (Goodpaster *et al*., 2006). This muscle atrophy, regardless of its causative pathomechanism, has been found to be strongly correlated to the development of OA. Since exercise increases muscle mass and improves muscle function, it is likely to play an important role in treatment and prevention of OA (Armin *et al*., 2009).

2.3.2 Pathology

OA is characterised by focal loss of cartilage with evidence of accompanying periarticular bone response. Clinically, it presents as joint pain and crepitus in the elderly age group (Souza, 2001), and is radiographically characterised by decreased joint space, osteophytes and a variety of deformities that develop as the disease progresses (Yochum and Rowe 1996; Marchiori, 1999; Taylor and Resnick, 2000).

2.3.2.1 Pathogenesis and Morphology

Normal hyaline cartilage comprises chondrocytes (1-2% of cartilage volume) embedded in extra-cellular matrix, which in turn is constituted by water, type-II collagen and proteoglycans (Young *et al*. 2006). The two main functions performed by articular cartilage, along with synovial fluid, include: reduced-friction movement within the joint, and, in weight-bearing joints, spreading the load across the joint to allow the underlying

bone to absorb shock and load (Levangie and Norkin, 2001; Standring, 2008). These functions require that cartilage be elastic and have a high tensile strength; which are attributes provided by proteoglycans and type-II collagen, both of which are produced by chondrocytes (Kumar *et al*., 2007). Articular cartilage constantly undergoes active degeneration and replacement. OA was previously thought to be a result of wear and tear, or an age-related degenerative process (Levangie and Norkin, 2001). However, it has since been shown that changes in the cartilage due to OA differ from those changes seen in the normal aging process, and result from any imbalance in normal chondrocytes regeneration and degradation (Kraus, 2007). The majority of pathological changes in OA occur in the cartilage, however, as the disease progresses, the bone and synovial structures also begin to show signs of degeneration (Marchiori, 1999; Resnick and Taylor, 2000; Kumar *et al*., 2007).

Cartilage Changes: Chondrocyte function can be affected by a variety of influences, including mechanical stresses, aging, metabolic and genetic factors, increased bone density and high oestrogen levels. Regardless of the inciting stimulus, early OA is marked by the degenerating cartilage containing more water and less proteoglycan (Kumar *et al*., 2007). This occurs as a result of an enzymatic degradation of the major structural components, aggrecan and collagen, which compromises the cartilage tensile strength and resilience, making it susceptible to load-bearing injuries (Boon *et al*., 2006).

Progression of these changes leads to breach of surface integrity, fissures, pitting, flaking of cartilage and development of vertical clefts (fibrillations), and a decrease in cartilage thickness. Cartilage loss is focal rather than widespread and usually restricted to the maximum load-bearing part of the joint (Boon *et al*., 2006). Gross examination at this stage reveals a soft granular articular cartilage surface (Kumar *et al*., 2007).

Bone Changes: As the cartilage fails in its load-transmitting function the bone immediately below is subject to increased pressure and responds by increasing its trabecular thickness (subchondral sclerosis), which in some cases reflects healed microfractures. The fracture gaps allow synovial fluid to be forced into the subchondral regions, forming fibrous walled cysts. At the margin of the joint there is formation of new fibrocartilage, which then undergoes endochondral ossification to form osteophytes. With severe cartilage loss, fissures may deepen and expose the subchondral bone to wear, with the unprotected bone ends becoming ivory-like due to thickening and vascularisation (eburnation), often with deep linear furrows (Boon *et al*., 2006). Small fractures can dislodge pieces of

cartilage and subchondral bone into the joint, forming loose bodies (joint mice) (Marchiori 1999; Taylor and Resnick 2000).

Other Changes: The synovium undergoes variable degrees of hyperplasia, sometimes as florid although less widespread as rheumatoid arthritis (RA). The outer capsule also thickens and contracts, usually retaining the stability of the remodelling joint. The muscles that act over the joint commonly show non-specific type-II fibre atrophy (Boon *et al.*, 2006).

2.3.2.2 Natural History

The course of OA is highly variable. Those patients with multiple affected joints tend to have a more rapid progression of OA in their individual joints (Felson, 1993). Advanced age and obesity are also associated with more rapid progression (Felson, 1993). Primary OA is generally regarded as slow in its progression, which is evident in one third to two thirds of radiographic OA cases. However, while it has been known to stabilise for many years, improvements are rare (Kumar *et al*., 2007). Symptomatic OA may progress, or improve, or may even be halted due to the fact that symptoms have been shown to be poorly correlated to radiographic progression (Kellgren, Lawrence and Bier, 1963). Osteophyte impingements on spinal foramina are a common cause of nerve root entrapment (Cramer and Darby, 2005; Morris, 2006), which may result in neurological deficits such as radicular pain, muscle atrophy or spasms, and sensory loss (Souza 2001; Morris, 2006). With time, total joint collapse may occur, but unlike RA, does not result in joint ankylosis (fusion) (Kumar *et al*., 2007).

2.3.2.3 Subsets of OA

Primary OA can be categorised into three major subsets, although it may not be easy to determine an exact distinction between the subsets (Doherty, 1994).

Nodal Generalised OA: Characterised by distal, and proximal but to a lesser extent, interphalangeal (IP) joint involvement; bony enlargement of distal interphalangeal (DIP) joint (known as Heberden's nodes), bony enlargement of proximal interphalangeal (PIP) joint (known as Bouchard's nodes) and familial clustering. This type of OA peaks at middle age and is common in females (Doherty, 1994; Boon *et al*., 2006).

Erosive OA: Characterised by involvement of the IP joints of the hands, often with florid inflammation and erosive changes, that later lead to deformities and ankylosis. A small proportion (15%) may evolve into seropositive RA (Doherty, 1994; Boon *et al*., 2006).

Isolated large joint OA:
Knee: This is the most common form of OA (Zhang *et al.*, 2008), often occurring bilaterally. It may involve predominantly the medial tibiofemoral, lateral tibiofemoral or patellofemoral compartment (Daniel, Akeson and O'Connor, 1990).

Hip: Predominantly involves the superior pole or the medial compartment of the hip (Parker and Pryor, 1993).

Spine: Apophyseal joint involvement is the only true form of OA that can involve the spine and is typically manifested by pain on extension on the spine. Intervertebral disc (IVD) degeneration with osteophyte formation is considered an integral part of OA, and commonly affects the lumbar and cervical regions. Diffuse intraosseeous skeletal hyperostosis (DISH) and ossification of posterior longitudinal ligaments (OPLL) are considered to be variants of spondylosis, and comprise flowing calcification of the disk margin and anterior and posterior longitudinal ligament, respectively (Doherty, 1994; Cramer and Darby, 2005; Morris, 2006).

2.3.3 Clinical Features

The development of signs and symptoms of OA may take many years after the onset of the disease to become clinically evident (Souza, 2001). This is due to the fact that the progression of the disease is highly variable and there tends to be a poor correlation to radiographic and microscopic progression (Yochum and Rowe, 1996; Marchiori, 1999; and Taylor and Resnick, 2000). Another possible reason that there may be a delay in the appearance of symptoms after degeneration has taken place is because of a lack of innervation within the cartilage (Lane and Buckwalter, 1993).

Joints commonly involved in OA are the knee, fingers, and spinal apophyseal joints. Less commonly are the hips, acromioclavicular and sternoclavicular joints, while carpal, elbow, ankle and glenohumeral joints are rarely involved in isolation (Taylor and Resnick, 2000; Boon *et al.*, 2006; and Kumar *et al*., 2007).

2.3.3.1 Signs and Symptoms

Symptoms of OA frequently have an insidious onset and an asymmetrical distribution, later becoming symmetric as the disease progresses (Roberts and Burch, 1996; Souza, 2001). Factors that may predict the presence of symptomatic OA and rate of progression include advanced age, obesity and multiple affected joints (as described in section 2.3.1.2).

The cardinal signs of clinical OA include bony swelling, synovial effusion, crepitus, restricted range of motion, joint deformity and, muscle weakness and atrophy (Gleberzon, 2001; Boon *et al.*, 2006). Symptoms associated with OA include pain, joint stiffness and functional impairment (Souza, 2008), although all signs and symptoms need not be present at the same time and to the same severity (Doherty, 1994). These are discussed as follows:

Pain: Pain usually begins as an intermittent localised deep ache in and around the affected joint, often exacerbated by movement. As the disease advances, pain may become more persistent, becoming present at night and during rest (Reid, 1992; Magee, 2008).

In knee OA, pain is usually localised to the anterior and medial aspects of the knee and upper thigh, commonly occurs with stair use, climbing in and out of vehicles, and doing daily activities such as bathing, standing from a seated position and sitting to use a toilet. These problems may be amplified in the presence of concomitant hip pathologies, where normal walking gait is likely to be altered as a result of the groin and leg pain (Reid, 1992; Magee, 2008).

Stiffness: Stiffness in the involved joints is typically present and worst first thing in the morning (morning stiffness) and lasts between five and thirty minutes. Stiffness may also be present later in the day after periods of rest or inactivity (gelling), but is brief and relieved by gentle movement (Reid, 1992; Magee, 2008).

This stiffness is often associated with impaired movement within the joint and may result from a number of causes: joint adhesion, capsular tightening and thickening, inflexibility of the overlying soft tissue and/or altered joint structure (e.g. as a result of osteophyte formation) (Yochum and Rowe, 1996).

Functional Impairment: The severity of functional impairment is largely dependent on the degree of degeneration, type of joint involved as well as the specific location of the degeneration within the joint. For example, if there is marked degeneration with osteophyte formation on adjacent articulating surfaces, which make contact during movement, one would expect to find that range of motion is impaired. Similarly, loose bodies may result in decreased range of motion as well as possible locking or buckling, especially if located within the knee joint. Crepitus develops as a result of cartilage loss and irregularities on articulating surfaces such that they no longer slide smoothly over one another (Gleberzon, 2001). This creates a stuttered-type motion, which can be palpated during range of motion examination. In severe cases, this crepitus may even create audible "pops". Crepitus is present in over 90% of patients with knee OA (Reid, 1992).

In severe cases, joint subluxation may occur when there is uneven wear of the joint surfaces. Over an extended period of time this will result in asymmetrical joint space narrowing and ultimately collapse (Taylor and Resnick, 2000). When this occurs in the knee, the medial tibiofemoral joint to typically more affected than the lateral (Marchiori, 1999) and in over 50% of patients will cause the development of a genu varus ("bow leg deformity") (Souza, 2001; Magee, 2008).

Functional impairment may also result not only from structural changes to the joint surfaces, but also from other changes associated with OA, such as arthrogenic muscle inhibition (Reid, 1992; Hopkins and Ingersoll, 2000). In this case, the patient may experience weakness due to atrophy of the surrounding muscles, as well as stiffness or decreased range of motion due to inflexibility (Hurley, 1998).

Table 2.4.2 Outline of Typical Symptoms of Osteoarthritis

Symptoms
• Patient over age of 45 • Insidious onset over months or years • Variable or intermittent pain over time • Mainly related to movement and weight-bearing, relieved by rest • Only brief morning (less than 15 minutes) stiffness and "gelling" (less than 1 minute) after rest • Usually only one or two joints painful (not multiple regional pain)
Signs
• Restricted movement (capsular thickening, blocking by osteophytes) • Palpable, sometimes audible, course crepitus (rough articular surfaces) • Bony swelling (osteophytes) around joint margins • Deformity, usually without instability • Joint-line or periarticular tenderness • Muscle weakness, wasting • No synovitis, or only mild if present (effusion, increased warmth)

(Adapted from Evans 2001; Boon *et al*., 2006)

2.3.3.2 Arthrogenic Muscle Inhibition (AMI)

AMI is defined as the failure of a functional muscle group to recruit all motor units during maximal voluntary contraction (Suter *et al*., 2000). When joint receptors are subjected to distension, compression, ligamentous stretch, effusion and pain, this protective mechanism is activated, causing reflex inhibition of the surrounding musculature (Crossman and Neary, 2005).

AMI appears to be most severe to in the acute stage of joint damage, in order to prevent further injury. However, pronounced levels of AMI may still be present months, and sometimes years after the initial joint damage (Rice and McNair, 2010). Following arthroscopic menisectomy, AMI was seen to result in 80-90% muscle inhibition in the first 24 hours, decreasing to 30-50% in the subsequent 2 weeks. Residual AMI (approximately 8% compared with healthy, age-matched controls) remain a mean of 4 years after joint injury, despite no radiological or clinical evidence of further degeneration (Becker *et al*., 2004).

In chronic conditions, such as OA, loss of output from articular sensory receptors occurs as a result of persistent swelling, inflammation, joint laxity, and structural damage. These are factors which have been identified as contributors to alter afferent input from the knee joint in patients with knee OA (Rice and McNair, 2010). This results in altered reflex pathways and decreased excitability of the quadriceps femoris muscle, with an inability to fully recruit the muscle fibres within the muscle group. The net results are:

1. Decrease muscle strength (actual and/or apparent weakness), causing delays in the rehabilitation progress (Hopkins and Ingersoll, 2000).

2. Altered movement patterns due to a change in motor control and joint proprioception, increasing the risk of pain, re-injury and accelerated degeneration because of the distorted joint forces (Reid, 1992; Bowers, 1996).

2.3.4 Diagnostic Criteria

Prior to the development of clinical criteria as proposed by Altman *et al*. (1986), diagnosis of OA was largely based on radiographic appearance and criteria proposed by Kellgren and Lawrence (1957).

Today the diagnosis of OA is primarily clinico-radiographic, that is both clinical and radiographic features are taken into consideration to determine the presence and severity of the disease (Gleberzon, 2001). It is widely acknowledged that radiographic changes may not be present in the early stages of degeneration, while only 40-50% of patients with radiographic evidence of OA are clinically asymptomatic (Roberts and Burch, 1996). For this reason, the American Rheumatism Association devised diagnostic criteria for OA in various joints, which are represented in Table 2.4.4, Table 2.4.5 and Table 2.4.6:

Table 2.4.4 Clinico-radiographic Classification Criteria for Osteoarthritis of the Knee

Traditional format	Classification tree format
1. Knee pain 2. Osteophytes ***In addition to*** 3. One of three: a. 50 years of age or older b. Stiffness less than 30 minutes c. Crepitus	Knee pain **And** osteophytes **Or** Knee pain **And** 40 years of age or older **And** morning stiffness greater than or equal to 30 minutes in duration **And** crepitus on motion

(Altman *et al*., 1986)

Table 2.4.5 Clinico-radiographic Classification Criteria for Osteoarthritis of the Hip

1. Hip pain
2. At least two of the following:
    a. ESR Westergreen less than 20mm/hour
    b. Radiographic femoral or acetabular osteophytes
    c. Radiographic joint space narrowing (superior, axial and/or medial)

ESR = Erythrocyte sedimentation rate

(Altman *et al*., 1991)

Table 2.4.6 Clinico-radiographic Classification Criteria for Osteoarthritis of the Hands

1. Hand pain, aching, or stiffness
2. Three or four of the following:
    a. Hard tissue enlargement of 2 or more of 10 selected joints*
    b. Hard tissue enlargement of 2 or more DIP joints
    c. Less than 3 swollen MCP joints
    d. Deformity of at least one of 10 selected joints*

** 10 selected joints are 2nd and 3rd DIP joint, 2nd and 3rd PIP joint, and 1st carpometacarpal joint*

DIP = Distal interphylangeal; PIP = Proximal interphylangeal; MCP = Metacarpophylangeal

(Altman *et al*., 1990)

2.3.4.1 Radiographic Diagnosis

There are eight cardinal signs of radiographic OA: asymmetric distribution, non-uniform loss of joint space, osteophytes, subchondral sclerosis, subchondral cysts, intra-articular loose bodies, intra-articular deformity, and joint subluxation (Taylor and Resnick, 2000). The radiographic presentation of OA varies depending on the joint involved, the anatomic relationships, and the stress to which the joint is subjected. Therefore, all eight signs need not be present in order to establish a diagnosis of OA; however they may be useful in determining the degree of underlying pathologic sequences involving the joint compartments. Table 2.4.7 describes the grading system used to establish radiographic severity of OA (Yochum and Rowe, 1996; Gleberzon, 2001).

Table 2.4.7 Kellgren-Lawrence Classification of Osteoarthritis

Classification	Description
Normal	No change
Grade I	Unlikely narrowing of the joint space, possible osteophytes
Grade II	Small osteophytes, possible narrowing of the joint
Grade III	Multiple, moderately sized osteophytes, definite joint space narrowing, some sclerotic areas, possible deformation of bone ends
Grade IV	Multiple large osteophytes, severe joint space narrowing, marked sclerosis and definite bony end deformity.

(Kellgren and Lawrence, 1957)

These eight cardinal signs are discussed as follows:

1) *Asymmetric Distribution:* There is frequently a visible disparity when comparing the extent of joint involvement with the unaffected (or lesser affected) joint on the contralateral side. The asymmetrical distribution of OA helps to distinguish it from inflammatory arthropathies, such as rheumatoid arthritis, which have a characteristically symmetrical involvement (Yochum and Rowe, 1996; Boon *et al.*, 2006).

2) *Non-Uniform Loss of Joint Space:* Reduction in joint space is most likely to occur at the regions of greatest intra-articular stress, which is especially evident in weight-bearing articulations such as the spine, hip, and knee (Yochum and Rowe, 1996; Taylor and Resnick, 2000**)**.

3) *Osteophytes:* Radiographically, these osteophytes are seen as bony outgrowths extending from the region of capsular insertion into the joint space. In very severe

cases the osteophyte may completely bridge the joint space, causing anyklosis of the joint (Yochum and Rowe, 1996; Marchiori 1999).

4) *Subchondral Sclerosis (Eburnation):* Trabecular thickening is usually evident in areas where there is the greatest loss in cartilage height. It occurs as a result of increased mechanical forces being transmitted to the joint surfaces that lack the shock absorbing effect of normal cartilage thickness. In order to counteract these increased forces, the existing trabecular bone thickens and new bone is formed. Subchondral sclerosis is seen on radiographs as increased areas of radio-opacity in the bone underlying regions of decreased joint space (Marchiori 1999; Taylor and Resnick 2000).

5) *Subchondral Cysts (Geodes):* These cysts are focal regions of loss in bone density, of variable size, which appear as rounded areas of radiolucency and often have a sclerotic margin. They are located in areas of previous subchondral sclerosis, and occur either as a result of synovial fluid intrusion through the exposed articular plate or secondary to trabecular fracture and subsequent necrosis (Marchiori 1999; Taylor and Resnick, 2000).

6) *Intra-Articular Loose Bodies (Joint Mice):* As joint degeneration progresses, flaking and fragmentation may result in intra-articular accumulation of free floating bodies, comprised largely of cartilage and occasionally subchondral bone (Yochum and Rowe, 1996; Marchiori, 1999).

7) *Articular Deformity:* Progressive deformation of the articular surfaces may occur following repetitive stress, causing large subchondral cysts, trabecular remodelling, fracture and collapse, which may be exacerbated my necrosis due to secondary vascular disturbances (Marchiori, 1999; Taylor and Resnick, 2000).

8) *Joint Subluxation:* The joint eventually becomes unstable and prone to displacement due to joint surface deformation, loss of joint space, and laxity within the structure of the ligaments and tendons. This alters in the load distribution, further increasing the unbalanced stresses of the joint, accelerating the degenerative process (Yochum and Rowe, 1996; Bergmann and Peterson, 2002).

2.3.4.2 Clinical Diagnosis

Altman *et al*. (1986) developed sets of criteria for the classification of idiopathic OA of the knee. This differs from prior criteria in that it uses classification trees, or algorithms, which serve to standardise and clarify the clinical definition of knee OA, and promote consistency in reporting of knee OA (Altman *et al*., 1986). The criteria in Table 2.4.8 were found to be 89% sensitive and 88% specific for diagnosis of knee OA.

Table 2.4.8 Classification Criteria for Diagnosis of Idiopathic Osteoarthritis of the Knee

Clinical and laboratory	Clinical and radiographic	Clinical **
Knee Pain + at least 5 of 9 below: Age > 50 years Stiffness < 30 minutes Crepitus Bony tenderness Bony Enlargement No palpable warmth ESR* < 40 mm/hr RF* < 1:40 SF OA*	Knee Pain + at least 1 of 3 below: Age > 50 years Stiffness < 30 minutes Crepitus + Osteophytes	Knee Pain + at least 3 of 6 below: Age > 50 years Stiffness < 30 minutes Crepitus Bony tenderness Bony Enlargement No palpable warmth
92% Sensitivity 75% Specific	91% Sensitivity 86% Specific	95% Sensitivity 69% Specific

** ESR = erythrocyte sedimentation rate (Westergren); RF = rheumatoid factor; SF OA = synovial fluid signs of OA (clear, viscous, or white blood cell count <2000/mm^3).*
*** Alternative would be 4 of 6, which is 84% sensitive and 89% specific.*
(Altman *et al*., 1986)

2.3.4.3 Differentiation from other Arthritic Diseases

Rheumatoid arthritis (RA): associated with more signs of inflammation involving the MCP, wrist, carpals and other peripheral joints, as well as the cervical spine (Cramer and Darby, 2005). Generalised OA involves the DIP, PIP and first CMC joints in the hand and, cervical and lumbar spine regions (Gleberzon, 2001). RA is distinguished from erosive OA through positive laboratory test, such as rheumatoid factor, ESR, and synovial fluid analysis (Ferri, 2004; Boon *et al.*, 2006).

Joints actively involved in rheumatoid arthritis seldom show osteophytes, therefore their presence is a useful indicator of OA if the patient presents with a mixed clinical picture (Gleberzon, 2001). If osteophytes precede rheumatoid involvement, it indicated that rheumatoid arthritis has evolved from an erosive OA. Conversely, they will only develop in secondary degeneration following RA burn out (Yochum and Rowe, 1996; Marchiori, 1999).

Pseudogout: differentiated from OA by presence of calcium pyrophosphate dihydrate (CPPD) crystals in synovial fluid, as well the in involvement of joints that are not typically associated with primary OA, such as the elbow and shoulder (McCarthy, 1998).

Table 2.4.9 Classification for Subsets of Idiopathic Osteoarthritis

Localised:
1. Hands: a. Heberden's and Bouchard's nodes (nodal) b. Erosive interphalangeal arthritis (non-nodal) c. Scaphometacarpal d. Scaphotrapezal 2. Feet: a. Hallux valgus b. Hallux rigidus c. Contracted toes (hammer/cock-up toes) d. Talonavicular 3. Knee: a. Medial compartment b. Lateral compartment c. Patellofemoral compartment 4. Hip: a. Eccentric (superior) b. Concentric (axial, medial) c. Diffuse (coxaesenilis) 5. Spine (particularly cervical and lumbar): a. Apophyseal b. Intervertebral (disc) c. Spondylosis (osteophytes) d. Ligamentous hyperostosis (DISH* or Forestier's disease) 6. Other single sites: a. Shoulder b. Temporomandibular c. Sacroiliac d. Ankle e. Wrist f. Acromioclavicular
Generalised (includes 3 or more sites listed above):
1. Small (peripheral) and spine 2. Large (central) and spine 3. Mixed (peripheral and central) and spine

** DISH = Diffuse Idiopathic Skeletal Hyperostosis*

(Altman, 1986; Yochum and Rowe, 1996; Marchiori, 1999; Taylor and Resnick, 2000)

2.4 Treatment Options

2.4.1 Pharmacologic Interventions

2.4.1.1 Medication

Analgesics: Among all analgesics, non-steroidal anti-inflammatory drugs (NSAIDs) are the most commonly prescribed drug for the treatment of pain and inflammation related to OA. However, their use is limited due to gastric and renal damage that can occur as complications following long-term use, particularly in the elderly (Gleberzon, 2001).

Recent studies have shown acetaminophen to be equally effective as other NSAIDs in relieving symptoms of OA, with the additional benefit of decreased gastric side effects (Bradley *et al*., 1991). Other medication such as meloxicam and celecoxib, are a newer form of NSAID that are selective cyclooxygenase-II (COX-2) inhibitors that have been shown to have less drug-related side-effects due to their increased selectivity and may soon replace non-selective NSAIDs as the drug of choice in OA treatment (Bjordal *et al*, 2004).

Topical analgesics, such as capsaicin cream, are a useful adjunct in the treatment of OA due to their ability to reduce pain and tenderness over the area of application. However, their absorption and ability to penetrate to deep sources of pain make them unreliable (McCleane, 2000).

Intra-articular steroids: Injections into the affected joint have been shown to produce marked improvement in symptoms; however, the relief they provide is somewhat short-lived, only lasting weeks to months, depending on the patient and area injected. This is problematic as regular high-dose corticosteroid injections are associated with decreased rate of repair and may even lead to further cartilage degeneration. It is for this reason that their use is limited to emergency situations and should not exceed 3-4 doses annually, with a minimum of 6 weeks between treatment of the same joint (Arroll and Goodyear-Smith, 2004).

Disease modifying agents: There are a number of pharmaceuticals available which are marketed as disease-modifying OA drugs (DMOADs), although there is little evidence to demonstrate that they have the ability to affect the natural history of the disease (Stagg, 2006). Amongst these are medication, such as 1) tetracycline, which are inhibitors of an enzyme linked to cartilage degeneration, 2) growth factor and cytokine manipulators and 3) genetic therapy (Sarzi-Puttini *et al*., 2005)

*Supplements: S*ulphated sugars, such as glucosamine sulphate, have shown some evidence in long-term studies to have significant disease modifying effect when administered either orally or intramuscularly. In a double-blinded placebo controlled trial, joint space narrowing and bone marrow lesions were significantly greater after one year in the placebo group as compared to those who received 800mg glucosamine sulphate daily (Wildi *et al., 2011*). Chondroitin sulphate is a predominant compound found in cartilage, the supplementing of which was found to have a significant pain relief over placebo, and similar cartilage sparing properties of glucosamine (Michel *et al*., 2005). However, those

receiving chondroitin sulphate alone or in combination with glucosamine showed no significant difference in pain relief over those receiving selective COX-2 inhibitor (celecoxib) (Clegg *et al*., 2006).

2.4.1.2 Surgery

Amongst the various surgeries available for OA are: loose body removal, osteotomy, arthrodesis, arthroplasty, synovectomy, debridement, tendon removal and nerve decompresson. Total knee and hip replacement have improved the quality of life and decreased morbidity due to OA, with over 90% of patients undergoing joint replacement surgery having good long term results. However, due the invasive nature of the procedure, it should only be considered at the end stage of joint disease, once all other options have been exhausted (Hochberg *et al*., 1995). Table 2.3.1 outlines the steps, as described by (Hochberg *et al.*, 1995), that medical professionals should follow when prescribing treatment for patients afflicted with OA of the hip or knee.

Table 2.5 Medical Management of Symptomatic OA of Hip and Knee

1. Non-pharmacologic therapy
2. *If effusion present, consider aspiration and injection of intra-articular steroids (e.g. triamcinolone hexacetonide)
3. Acetaminophen (up to 1mg qid) for pain and symptom control
4. *If necessary, add topical capsaicin cream
5. If response inadequate, use alternative analgesic, low dose ibuprofen (up to 400mg qid) or non-acetylated salicylates
6. If response inadequate, use full-dose NSAID (with misoprostol if patient has risk factor for upper gastro-intestinal bleeding or ulcer disease)
7. If response is inadequate and surgery is contraindicated, consider referral for joint lavage and/or arthroscopic debridement
8. If response inadequate, and surgery is not contraindicated, consider referral for joint surgery (osteotomy, total joint arthroplasty)

**Applicable for knee joint only*

qid = four times daily

(Hochberg *et al*., 1995)

2.4.2 Non-pharmacologic Interventions

2.4.2.1 Rehabilitation

The main goals of exercise therapy are to reduce pain, improve joint function and increase stability (Kendall *et al.,* 2005; Sahrmann, 2010). Rehabilitation and physiotherapy are frequently prescribed to counteracting the muscle weakness and atrophy which has contributed to the disease process (Slemenda *et al*., 1998). However, in order to adequately counteract these processes, exercise routines must be performed on a regular and continuous basis; focusing on either muscle stretching and strengthening activities, or

aerobic training. These exercises can either be performed on land (Brosseau *et al*., 2003) or in water (Lund *et al*., 2008; Cadmus *et al*., 2010).

Aquatic exercises have been shown to be less effective than land-based exercises in relieving symptoms of OA (Lund *et al*., 2008), except in obese patients who benefitted from being able to perform relatively pain-free movement as a result of increased buoyancy and decreased joint loading in the water (Cadmus *et al*., 2010). Studies have also showed that both high and low-intensity aerobic exercises are beneficial in patients with OA (Brosseau *et al*., 2003), and patients should, therefore, have exercise routines customised to cater for their particular fitness and ability.

Jan *et al*. (2008) found pain relief and improved joint function to follow treatment with both low- and high-resistance training. Although high-resistance training appeared to yield greater improvements in joint function and pain reduction, the difference between groups was not statistically significant. Jan *et al*. (2008) speculated that resistance training (e.g. walking on uneven terrain) may increase balance and proprioception. Additionally, increased muscular strength, decreased pain and improved joint position sense were found to be greater in those performing weight-bearing exercises, as compared to non-weight-bearing exercises (Jan *et al*., 2009).

There is a paucity of information to support the benefits of specific sports in the prevention and treatment of OA. However, muscle weakness and atrophy have been reported to occur prior to the onset of symptomatic OA (Slemenda *et al*., 1997) and in a long-term study by Armin *et al*. (2009), strong quadriceps muscles appeared to have a protective effect on the joints, which showed less cartilage loss in the lateral and patellofemoral compartments of the knee. This result supports findings from an animal study (Otterness *et al*., 1998) in which signs of joint degeneration (reduced proteoglycan content and synovial fluid volume) was found in a group of sedentary hamsters, whereas the exercising group showed smooth articular surfaces after three months of daily activity (Otterness *et al*., 1998). Exercise was also found to have a beneficial effect on human cartilage in a study conducted on participants who had undergone a partial medial meniscectomy three to five years prior to commencement of the exercise programme (Roos and Dahlberg, 2005). After four months of exercise therapy the treatment group showed not only increased activity levels, but also raised glucosaminoglycan levels as compared to the control group, who had no alteration to their daily level of activity.

An argument could therefore be made that physical activity not only stimulates muscle strength and joint stability but also makes cartilage less prone to degenerative change. It is speculated that the chondroprotective property may be due to the release of interleukin-10 (IL-10) into the synovial fluid following acute resistance exercise in patients with knee OA (Helmark *et al*., 2010). IL-10 is a known anti-inflammatory cytokine, which suppressed the release of macrophage inflammatory cytokines as well as stimulates activity in chondrocytes and synoviocytes, thereby interfering with the pathogenesis of OA (Hart *et al*., 1995).

McCarthy *et al*. (2004) found that the supplementation of an at-home exercise programme with class-based exercises significantly increased mobility and decreased walking pain in patients with knee OA. This highlights the importance of patients undertaking supervised exercises as part of their exercise prescription; since a dose response between increased adherence and benefit has previously been identified (van Baar *et al*., 2001), one could argue that the lack of improvement seen in the at-home group was due to poor compliance.

Van Baar *et al*. (1998) conducted a clinical trial to determine the effect of exercise therapy on patients with OA of the hip and knee in terms of pain, observed disability and medication use. Participants in the intervention group showed noticeable improvement in their ability to perform various functional movements and decreased pain (recorded by means of visual analog scale (VAS)), as well as beneficial effect on muscle strength of the hip, reduction in paracetamol use, and global effect as perceived by the patient. Exercise did not appear to have an effect on the use of NSAIDs, self-reported disability, or active range of motion.

However, at six and nine months, these benefits were seen to decline and ultimately measurements taken at the final assessment resulted in a similar clinical status as the non-intervention group (van Baar *et al*., 2001). This suggests that exercise may have a small to moderate effect on pain and disability in individuals with OA of the hip and/or knee (van Baar *et al*., 1998). However, in order to retain the beneficial effects, Sluijs and Knibbe (1991) state that measures must be taken by both the patient and practitioner to ensure long-term compliance.

Fransen and McConnell (2008) found, in a systemic review, that land-based therapeutic exercise only offered short-term relief. The small effect size of exercise was comparable to reported estimates for NSAIDs.

Fitzgerald (2005) suggested that the benefit of exercise for knee OA was limited to a small to moderate effect, due to the limitations in the design and implementation of the exercise programmes. Fitzgerald (2005) stated that the same factors that have been found to influence the degree of disability and/or progression of the disease may also influence the patient's ability to participate in and, to a certain extent, vary their response to a rehabilitation protocol. Quadriceps inhibition or activation failure, obesity, passive knee laxity, knee misalignment, fear of physical activity and self-efficacy were identified as such factors (Cooper *et al*., 2000). Additionally, persistent impairments at lower extremity joints other than the knee were suspected to alter the overall functional improvement to rehabilitation because of the inability of that impaired joint to compensate for the knee pathology. Jadelis *et al*. (2001) found that when testing dynamic balance in individuals with weak knee muscles, those with stronger ankle musculature were better able to compensate for the weakness than those with additional ankle weakness. This finding outlines the importance of addressing strength in all muscle groups of the lower limb, as opposed to isolated knee exercises, to maximise responsiveness to rehabilitation.

Sturnieks, St George and Lord, (2008) suggested that the Otago Exercise Programme, a combination of strength and balance exercises, would be most beneficial in reducing the risk of fall in the elderly.

It has been found that simple knee flexion and extension exercises over a period of time are sufficient to improve joint function and muscle strength. However, when these exercises are performed while weight-bearing, there is the additional benefit of improved joint position sense (Jan *et al*., 2009).

There appears to be little consensus as to which form of rehabilitative therapy is most effective for improving knee OA symptoms and functional impairment. While cardiovascular training appears to benefit OA by reducing stress on the knee joint as a result of weight loss, strength training is useful in ensuring that the knee musculature is properly conditioned to cope with the forces applied to it.

2.4.2.2 Manual and Manipulative Therapy (MMT)

Gatterman (2005; Haldeman, 2005) defined manual therapy as the therapeutic application of manual force, including all procedures in which the hands are used to mobilise, adjust,

manipulate, apply traction, massage, stimulate, or otherwise directly or indirectly influence the patient's function and/or health.

Chiropractic adjustment was described as a specific form of articular manipulation characterised by direct (short lever), specific, high-velocity, low amplitude thrust, usually associated with restored range of motion, pain-free mobility and function following its application (Bergmann and Peterson, 2002). It is, therefore, theorised that if knee OA can be caused or aggravated by a loss of joint mobility, then manipulation could be used to as a primary treatment in order to restore normal joint mechanics, thereby improving the patient's symptoms of stiffness, joint hypomobility, chronic pain and joint impairment (Hertling and Kessler, 2006).

Manipulation is also reported to reduce arthrogenic muscle inhibition by stimulating joint receptors (Wyke receptors), resulting in pain inhibition and reduction of the arthrogenic muscle inhibition. This pain inhibition occurrence is explained by the gait control theory, in which noxious impulses and "substance-P" release is blocked by the stimulation of mechanoreceptors (Melzack and Wall, 1965)

Tucker, Brantingham and Myburg (2003) conducted a clinical trial to determine the relative effectiveness of knee joint manipulation and oral NSAIDs over a period of three weeks. While no statistically significant difference was noted between the treatment groups, intra-group comparison was favourable of manipulation for improved range of motion and pressure-pain threshold. Although the results of this study indicate that manipulation and NSAIDs are equally effective in the short-term treatment of knee OA, Tucker, Brantingham and Myburg (2003) believed that SMT would have greater long-term results as it acts to restore joint function, prevent pain and reduce inflammation. NSAIDs may have similar effect on the patient, however they fail to address the cause of inflammation and the altered biomechanics, which in the long-term may result in overuse of the knee due to loss of pain-protecting mechanisms (Hertling and Kessler, 2006). Additionally, it was recommended that use of NSAIDs be restricted to episodes of acute inflammation due to the numerous complications of long-term administration (Gottlieb, 1997).

Fish *et al*. (2008) later investigated the relative effectiveness of knee joint mobilisation and capsaicin cream for knee OA. Capsaicin cream has previously been demonstrated to be a superior alternative to a placebo in the treatment of painful disorders such as knee OA and general osteoarthritis (Gemmell, Jacobson, and Hayes, 2003). Fish *et al*. (2008) was not able to demonstrate a statistically significant difference between the results of the

treatment groups, with both showing a favourable response. Based on the results of Tucker, Brantingham and Myburg (2003) and Fish *et al*. (2008), it appears as though both manipulation and mobilisation of the knee joint are more effective than placebo in the treatment of knee OA.

Pollard *et al*. (2008) found that not only was a MMT protocol, consisting of soft tissue mobilisation and an impulse thrust applied to the knee joint complex, beneficial in reducing symptoms of patellofemoral pain syndrome (PFPS), such as pain, reduced range of motion, crepitus and impaired function. But, a significant change in hip range of motion was noted in the treatment group receiving knee manipulation as compared to the placebo-control group, who were administered interferential current set at zero. Pollard *et al*. (2008) attributed this change to the effects of a single joint manipulation on the biomechanics of the FKC.

Furthermore, there have been studies which suggest that correcting a biomechanical abnormality in the in the hip (Cliborne *et al*., 2004; Currier *et al*., 2007) and lumbopelvic (Iverson *et al*., 2008) regions would have a favourable outcome on knee joint pain and dysfunction secondary to PFPS or knee OA. Currier *et al*. (2007) and Iverson *et al*. (2008) went on to develop sets of Clinical Predictive Rules (CPRs) to help identify which patients with knee pathologies would demonstrate a favourable short-term response to MMT of adjacent joints along the kinetic chain (i.e. the hip and lumbopelvic spine, respectively).

Cliborne *et al*. (2004) performed four clinical hip tests (Functional squat, FABER, hip flexion and hip scour tests) on patients with knee OA as well as an asymptomatic control group, with pain and range of motion responses being the dependent variables. It was found that significantly more patients in the knee OA group had pain during the hip tests, as compared to the asymptomatic group. Hip mobilisation was administered based on the presence of painful clinical test findings, with a significant short-term reduction in pain and increase in range of motion being noted following a single intervention session. Cliborne *et al*. (2004) suspected that the increased prevalence of painful hip test findings may be due to alterations in gait and knee function as a result of the underlying pathology, and thus recommended that the hip region be evaluated for impairments which may contribute to the symptoms a knee OA patient may experience.

Following research by Cliborne *et al*. (2004), Currier *et al*. (2007) sought to develop a set of CPRs which would identify which patients with knee OA and knee pain would best respond to hip mobilisation. Currier *et al*. (2007) identified five variables (hip or groin pain

or paraesthesia; anterior thigh pain; passive knee flexion less than 122°; passive hip medial (internal) rotation less than 17°; and pain with hip distraction) which would increase the probability of successful treatment from 68% to 92% if one variable was present, and to 97% if two variables were present.

Similarly, Iverson *et al*. (2008) developed a set a CPRs to predict an immediate successful response in patients with PFPS to lumbopelvic manipulation, including side-to-side difference in hip internal rotation >14°, ankle dorsiflexion >16°, navicular drop >3mm, no self-reported stiffness when sitting >20min, and squatting reported as most painful activity. Based on the results of this study, the strongest predictor of treatment success was side-to-side hip internal rotation asymmetry >14°. Iverson *et al*. (2008) suggested that this may in part be due to the concept of regional interdependence, whereby an impairment or dysfunction in a remote region may affect or contribute to a patient's primary complaint (Deyle *et al*., 2000; Cibulka and Threlkeld-Watkins, 2005; Cleland *et al*., 2007). In this case, it suggests that alterations in joint mobility of the lumbar spine, sacroiliac joint, or hip may contribute to the development of PFPS and other knee pathologies.

Although both MMT and rehabilitation have been found to be beneficial for knee OA, there have been few studies to investigate the effectiveness of combined treatment. Deyle *et al*. (2000) showed that rehabilitation combined with MMT to the full lower kinetic chain was more effective than a placebo in improving function, and reducing pain and stiffness. The four week treatment period treatment consisted of daily range of motion, strengthening and cardiovascular exercises, as well as mobilisation applied twice weekly to the knee, and to the lumbar spine, hip and ankle. On average, the intervention group had increased their baseline WOMAC scores by 55.8% and six-minute walking distance by 13.1%, which was seen as clinically and statistically significant. At one-year post-treatment, patients in the intervention group reported fewer intra-articular steroid use or total knee arthroplasty, and outcomes measures indicated persistent beneficial effects of treatment. Based on this information, the authors concluded that combined MMT and rehabilitation can provide long-term benefits for patients with knee OA, and potentially offset the need for surgical interventions (Deyle *et al*., 2000).

Deyle *et al*. (2005) used similar methodologies to compare the effectiveness of a supervised clinic-based physical therapy programme with a home-based exercise programme. All participants were given identical at-home exercise programmes, while those receiving in-clinic treatments had additional supervised exercises as well as FKC MMT. Both groups measured significant reduction in WOMAC scores and increases in the

six-minute walk test distance. However, the clinic-bases treatment group's improvements were almost double that of the at-home exercise only group. Delye *et al.* (2005) attributed this difference in the two groups to the application of MMT and supervised exercises, which was in keeping with the methodology used in their previous study (Deyle *et al*., 2000) that produced similar results. Due to the number of variables that changed between intervention groups, one cannot be certain whether it was the additional application of MMT or supervised exercises, or indeed the combination thereof, which produced the superior results. One can, however, conclude, based on the fact that the difference between groups was not sustained past eight weeks, that the continued application of clinical therapy was necessary to have a long-term results.

2.5 Conclusion

There is a growing body of evidence to support the benefit of exercise and manual therapies in the management of knee OA. Although not curative, both types of treatment appear to yield positive results with regard to reduction of the major symptoms (pain, stiffness and dysfunction), and best evidence would support the combination of rehabilitation and MMT of the FKC. However, more research is needed to determine the effectiveness of FKC manipulation alone, in light of the fact that patient compliance to an exercise programme is often inadequate.

Therefore, this research is needed to further investigate a combination of a full kinetic chain MMT and rehabilitative protocol, and its individual components to determine their relative effectiveness.

CHAPTER THREE

METHODOLOGY AND MATERIALS

3.1 Introduction

This chapter includes a detailed description of the study design, individuals selected to participate in the study and the intervention they received once included. The methods of evaluation used in the study as well as the statistical procedures used in the analysis of the data are also discussed.

3.2 Study Design

This study was designed as a single-blinded, randomised comparative clinical trial in which convenience sampling was utilised (Brink, 2006). This study was conducted in order to determine whether a combination of full kinetic chain (FKC) manual and manipulative therapy (MMT) and rehabilitation was more beneficial than FKC manipulation or FKC rehabilitation alone in the management of patients with osteoarthritis of the knee.

This study design was approved by the Faculty of Health Sciences Research and Ethics Committee (FHSEC 031/10) [Appendix R] indicating that the research protocol satisfied the ethical requirements set out by the committee for such studies. Furthermore, this approval indicates that the research protocol is in line with the Declaration of Helsinki, 1975 (Johnson, 2005).

3.3 Method

3.3.1 Advertising

Participants were recruited by the researcher, personally approaching retirement homes throughout the greater Durban region, as well as by placing advertisements on notice boards at the Durban University of Technology (DUT), community halls, shopping centres and places of worship [Appendix A]. Verbal permission was sought for distribution of pamphlets, flyers or posters at sites outside of the DUT.

3.3.2 Sample

3.3.2.1 Sample Size

The individuals who responded to the advertisement were screened and accepted based on the inclusion and exclusion criteria. The sample group for this study consisted of sixty-six participants. The sample was stratified into two age groups (38-59 and 60-80), prior to treatment allocation, to ensure that all treatment groups were equally representative of all age levels.

3.3.2.2 Sample Allocation

Once accepted into the study, patients were randomly allocated into three treatment groups using a Randomised Allocation Chart [Appendix Q]. For statistical purposes, the groups were named Group A - Manipulation, B - Rehabilitation, and C - Combination. Group A received FKC manual and manipulative therapy (MMT) only, Group B received FKC rehabilitation only, and Group C received FKC MMT combined with FKC rehabilitation.

3.3.3 Sample Characteristics

If the respondent met the criteria for the study, a consultation was booked, at which point they were presented with a Letter of Information and Informed Consent form [Appendix I], which they were required to sign. The following inclusion and exclusion criteria were assessed using a case history [Appendix B], physical examination [Appendix C], lumbar and pelvis [Appendix D], hip [Appendix E], knee [Appendix F] and, foot and ankle [Appendix G] regional examinations.

3.3.3.1 Inclusion Criteria

These included the criteria developed and discussed by Altman *et al.* (1991) and Kellgren and Lawrence (1957).

A. Criteria, as developed by Altman *et al.* (1991), required a minimum of one of the three clinical criteria below for diagnosis of knee OA.

1. Knee pain and crepitus with active motion and morning stiffness ≤ 30 minutes (with age 38 ≤ 80 years of age).

2. Knee pain and crepitus with active motion and morning stiffness >30 minutes and bony enlargement (with age 38 ≤ 80 years of age).

3. Knee pain and no crepitus and bony enlargement (with age 38 ≤ 80 years of age).

B. The following three criteria were all required:

4. Knee pain of ≥ 1 year duration and able to stand and walk without severe varus/valgus deformity and/or severe instability (Kellgren and Lawrence, 1957).
    - Severe instability in this study was defined as greater than 5-10° increased ROM in the "anterior drawer" tests, or a varus/valgus stress test as compared to the opposite knee (Magee, 2008).

5. Respondents required a score of ≥ 500mm out of a total of 2400mm (or ≥ 21%) on the WOMAC scale to be included (Yeomans, 2000).

6. Respondents could not have a history of meniscal or other knee surgery in the past six months (Pollard *et al.,* 2008).

7. All respondents were required to read and sign the Letter of Information and Informed Consent form [Appendix I].

3.3.3.2 Exclusion Criteria

Respondents were excluded if they had:

1. Significant visual disorders, severe vestibular disorders, neurological and peripheral sensory disorders which may have constituted a contra-indication to exercise (Boon *et al*., 2006).

2. A history of knee or hip joint replacement, severe varus or valgus deformity, instability, fracture and severe osteoporosis, rheumatoid arthritis, or frank avascular necrosis with or without moderate or severe deformity (Yochum and Rowe, 1996).

3. A history of significant lumbar herniated disc injury with sequelae, such as signs or nerve root compression (Iverson *et al*, 2008) and low back pain radiating to or below the level of the knee (Morris, 2006: Currier *et al*, 2007).

4. Severe balance and proprioception problems (i.e. inability to stand with and/or without marked spinal or hip deformity) (Crossman and Neary, 2005; Boon *et al*., 2006).

5. Long-term chronicity of knee OA combined with multiple treatment failure – especially multiple failure with previous physical treatment (≥ three), with and/or long-term severe pain, and/or a severely complicated or complex disorder (such as multiple co-morbidities combined with knee OA such as a mix of: knee, hip and lumbosacral OA, and/or cardiovascular and/or auto-immune disease), or a severely disabled and/or a patient with severe and decreased functional ability and/or severe clinical depression.

The reason these respondents were excluded is because they would have been considered preferable candidates for surgical intervention (Daniel, Akeson and O'Connor, 1990).

The following statements (i-iv) need to be taken into account when reading exclusion criterion number five:

I. Pain: If the patient gave a history that could be interpreted as having stayed constantly or chronically at a high level of an estimated verbal analogue score (VAS) of ≥ 7 or WOMAC score of 1680-1920mm (70-80%), out of a maximum worst score of 2400mm, for three to five years or longer.

II. Complicated or complex: Three or more disorders at one time in the same patient (with knee OA) as listed from number 1-4 of the exclusion criteria.

III. Severely disabled: dependent on a cane, brace or walker 75 to 100% of the time when ambulating; severe cardiovascular disease; severe instability in the knee or other joints or possibly less than, or markedly less than half the normal ROM.

IV. Clinically depressed: determined by history and use of the Beck Depression Inventory (BDI). The BDI has been validated for measuring depression in clinical and non-clinical settings (Beck *et al.*, 1961). Based on this outcome, participants were excluded if their baseline was score or 21 or greater.

6. Patients were permitted to continue taking any medication that they had initiated more than six weeks prior to joining this research (Poul *et al.,* 1993; Seth and Seth 2009). Any new medication that they began during the course of this research would have resulted in their exclusion as it may have been the particular drug that caused their improvement or deterioration, and not the research trial itself**.**

7. Patients were not included if they had received cortisone injection/s to the knee within 30 days prior to the start of the research (Clibourne *et al*., 2004).

3.4 Clinical Procedure

3.4.1 Participant Assessment

Respondents were required to contact the Durban University of Technology Chiropractic Day Clinic (DUT CDC) telephonically to determine whether they met the requirements of the study. This was established by asking the patient the following questions:

Table 3.1 List of Questions and Required Responses during Telephonic Interview

	Questions asked	Required response for inclusion
1	Are you between the ages of 38 and 80?	Yes
2	Have you had knee pain for longer than 1 year?	Yes
3	Do you have a history of trauma or surgery to the lumbar spine or lower limb?	No (If yes, not in the last 6 months or with significant side effects)
4	Are you able to stand and walk on your own, with minimal need and/or without significant dependence on a cane or walker?	Yes
5	Do you suffer from a chronic medical condition that would require you to take regular medication?	No (If yes, not having started in the last 6 weeks)
6	Would you be prepared to have radiographs taken of your lower limb?	Yes

If the respondents appropriately answered the above six questions, an initial consultation was scheduled at the DUT CDC. This consultation included further participant screening by means of a brief case history (Appendix B) and examination of the cardiovascular, respiratory, abdominal and neurological systems (Appendix C) for potential pathologies that would have resulted in their exclusion from the study. Thorough examination of the lumbar spine and pelvis (Appendix D), hip (Appendix E), knee (Appendix F), foot and

ankle (Appendix G) regions was also performed, with special consideration to the following orthopaedic indications:

3.4.1.1 Indications for the Application of FKC Manual Therapy

3.4.1.1.1 The Lumbopelvic Spine

A study on Patellofemoral Pain Syndrome (PFPS) suggested that the strongest predictor of whether manipulation of the lumbopelvic spine would decrease knee pain was a side-to-side difference in hip internal rotation range of motion greater than 14° (Iverson *et al.*, 2008).

3.4.1.1.2 The Hip Joint

Cliborne *et al*. (2004) demonstrated that the presence of hip pain and pain on squatting, as well as restricted hip flexion and/or a positive scouring test predicted a favourable response to hip mobilisation in terms of knee OA outcomes.

Deyle *et al.* (2005) demonstrated that people with the worst Kellgren-Lawrence scores (i.e. people with the most severe knee OA degenerative findings per x-ray) gained added patient relief or better knee OA outcome measures after receiving hip flexion and extension mobilisation.

Currier *et al*. (2007) further suggested that, the following findings predicted a better outcome for knee OA if hip mobilisations were applied when patients presented with:

(1) Hip or groin pain or paraesthesia,
(2) Anterior thigh pain,
(3) Passive knee flexion less than 122°,
(4) Passive hip medial (internal) rotation less than 17°, and
(5) Pain with hip distraction.

3.4.1.1.3 The Knee Joint

In terms of MMT to the knee, Deyle *et al.* (2005) suggested that, in most instances, the introduction of knee extension mobilisation early on in knee OA care yielded a fast response (in terms of decreased pain and increased mobility) with minimal concern or

adverse side effects. Deyle *et al*. (2005), however, cautioned against the early introduction of flexion mobilisation (Grades 3-5), especially in the presence of pain to provocation in flexion and/or marked restriction.

It was recommended that the Macquarie Injury Management Group (MIMG) Knee Protocol, developed by Pollard *et al*. (2008), be used to decrease the tenderness often elicited by patella mobilisation. This MIMG protocol used a combination of passive patella mobilisation combined with active knee extension to better decrease discomfort and more effectively increase patellar glide in knee OA patients.

3.4.1.1.4 The Ankle Joint

Earlier research by O'Brien and Vicenzino (1998) and Collins, Teys and Vicenzino (2004) suggested that: (1) lack of dorsiflexion range of motion and/or (2) lack of motion palpation end feel spring and/or (3) lack of anterior to posterior talar glide may be predictors of good response to ankle manipulation. An ankle joint with less than 16° dorsiflexion range of motion, when the knee is flexed, was predicted by Iverson *et al.* (2008) to be a good indication for manipulation to the ankle joint to help decrease knee pain in PFPS.

3.4.1.1.5 The Joints of the Foot

The following were found to be good indicators for a positive response to first metatarsophalangeal joint (MTPJ) manipulation in for the treatment of knee OA (Brantingham *et al*., 2007):

- Less than 65° dorsiflexion ROM; and/or
- Loss of good quality motion and motion palpation end feel spring; or
- Decreased axial elongation per the first MTPJ.

3.4.1.2 Radiographic Assessment

Although diagnosis of knee OA was made primarily through a clinical examination, knee radiographs were taken of respondents who qualified and consented to participate in the clinical trial. The purpose of the radiographs was to determine the grade of osteoarthritic change, according to the Kellgren-Lawrence scale (Kellgren and Lawrence, 1957).

Additionally, the radiographs were instrumental in confirming suspicions of contra-indications to treatment, and in ruling out other pathologies that would have resulted in their exclusion from the study. In addition to knee radiographs, the respondent's history and physical examination may have indicated a need for lumbosacral/pelvic, hip, ankle and/or foot radiograph/s (see exclusion criteria 2, 3, and 5– Section 3.3.3.2).

3.4.2 Interventions

All respondents in this study were given a medication diary [Appendix O], which they were required to complete daily in order to monitor any changes in frequency and dosage of analgesic medication consumed.

3.4.2.1 Manipulative Therapy

Manipulative therapy to the knee focused on restoring knee flexion and extension by lesser grades of mobilisation as recommended by Deyle *et al.*, (2005) and Fish *et al.*, (2008). Treatment also involved patellar mobilisation as per Pollard *et al.*, (2008), along with careful high velocity low amplitude (HVLA) axial elongation of the knee joint as recommended by Fish *et al.,* (2008).

Additionally, MMT was applied where needed to the full kinetic chain (any indicated axial or appendicular joint dysfunction; as found in the lumbar spine, pelvis, hip, ankle, and foot) using other diversified techniques, such as HVLA manipulation or mobilisation as outlined in Schafer and Faye (1989), and/or Bergmann and Peterson (2002). Also, the hip technique, as outlined by Hoeksma *et al.* (2004) and the use of HVLA knee manipulation methods from Tucker, Brantingham and Myberg (2003) were utilised when indicated.

The particular joint dysfunction also known as the subluxation complex or manipulable lesion (Bergmann and Peterson, 2002; Byfield, 2005) was chosen based upon findings in the regional examinations [Appendices D-G].

3.4.2.2 Rehabilitation

Rehabilitative therapy included exercises, focused soft tissue treatment and stretch to the knee musculature and elsewhere along the full kinetic chain where needed, based upon the functional assessment performed during the initial consultation (Deyle *et al.*, 2005). Also included in rehabilitation were patient advice, education and home exercise recommendations for managing their knee OA.

The rehabilitation protocol was standardised across Groups B and C, with minor case by case variations (Group B received home exercise instruction and a follow-up check before the fourth visit but no soft tissue treatment as recommended by Deyle *et al.* 2005). All participants receiving rehabilitation were given a brochure [Appendix P] which outlined the protocol which they were required to follow.

An exercise diary [Appendix O] was kept by all patients in the rehabilitation groups (*n*=44) to monitor at-home exercise compliance.

3.5 Intervention Frequency

- At the initial treatment, all patients in Groups B and C received training in a rehabilitation programme, which they were required to complete on non-treatment days.
- Groups A and C were required to return for a further five compulsory treatments in the first three weeks (twice per week for treatment).
- Group B were only required to return for the fourth treatment/training session, with the second, third, fifth and sixth visits considered optional.

All groups were required to return to the clinic within one week after the sixth treatment to have follow-up readings taken.

Table 3.2 Outline of the Treatment Protocols for Each Group.

	Group A Manipulation only	Group B Rehabilitation only	Group C Combination
Number or mandatory visits	6 + one week follow-up	2 + one week follow-up	6 + one week follow-up
Manipulation/mobilisation	Yes	No	Yes
Soft tissue manipulation	No	No	Yes
Stretching exercises	No	Yes	Yes
Strengthening exercises	No	Yes	Yes
Range of motion exercises	No	Yes	Yes
Medication diary	Yes	Yes	Yes
Rehabilitation diary	No	Yes	Yes

3.6 Measurement Tools

3.6.1 Subjective Data

Subjective data was obtained from the following:

3.6.1.1 Western Ontario and McMaster Universities Osteoarthritis Index

The Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) [Appendix J] has been utilised to detect changes in function and quality of life in patients suffering from knee OA using multiple questions with a visual analogy scale (VAS).

The WOMAC has been shown to be a valid and reliable tool that has been broadly and frequently utilised to measure knee and hip OA severity, thus allowing comparison to a large number of studies and trials (McConnell, Kolopack and Davis, 2001).

For the purposes of this study, successful clinical change was defined as a minimum 20% change. In addition, a respondent's WOMAC score of ≥ 500mm (or at least 21% out of a maximum of 2400mm) was required for inclusion (Barr *et al*., 1994).

3.6.1.2 The McMaster Overall Therapy Effectiveness Tool

Chan *et al.* (2006) indicated that the McMaster Overall Therapy Effectiveness (OTE) Tool [Appendix K] is a valid and reliable questionnaire that allows the patient to classify the change in their health status: whether their knee OA symptoms or overall quality of life has improved, remained the same, or deteriorated since the last visit.

A positive 30% (2.1 point) change (out of 7) was considered a successful, statistically and clinically significant change.

3.6.1.3 Beck Depression Inventory

The Beck Depression Inventory (BDI) [Appendix L] comprises 21 questions which assess depression-specific symptoms in order to determine the intensity, severity, and depth of depression (Beck, Steer and Brown, 1996).

For the general population, a score of 21 or greater represents a strong indication of depression. Based on this score, participants were excluded from this study if their baseline score was greater than 21.

3.6.1.4 Berg Balance Scale

The Berg Balance Scale (BBS) [Appendix M] was developed to measure risk of falls among older people with impairment in balance function by assessing the performance of 14 functional tasks (Berg *et al*., 1989). The BBS scores patients from 0 to 4 on each item for a maximum of 56, with a score of 45 or lower representing increased risk of a fall (Lajoie and Gallagher, 2004). A 6 point change in BBS score is considered a clinically significant decrease in risk of a fall (Stevenson, 2001).

The BBS begins with the timed One Legged Standing Test (OLST), which the patient must fail (be unable to stand for a period of ≥ 10 seconds on one leg) in order to proceed to the remaining 13 functional tasks of the BBS test (Hawk *et al.*, 2006).

Knee OA patients who failed the OLST at the initial visit were monitored as an impaired balance subgroup (with an OLST and BBS) at all clinic assessments, even if the patient went on to pass the initial BBS (Gleberzon, 2001).

3.6.2 Objective Data

3.6.2.1 Knee range of motion

Inclinometer readings were taken for knee flexion and extension only, to evaluate the patient's knee range of motion (ROM), and recorded in a table [Appendix N]. A clinically significant increase in ROM was indicated by 5-10° of improved movement (Evans, 2001). The instrument used in this study was the Saunders Digital Inclinometer (The Saunders Group Inc., 1998).

3.6.2.2 One Legged Standing Test (OLST)

The OLST is a standalone orthopaedic test for balance (Berg *et al*., 1992) but it also forms a part of the BBS (one of the 14 functional tasks). It was performed on all patients at the initial consultation, but only at a follow-up appointment for participants who formed the

impaired balance subgroup. A clinically significant result was considered if the participant's recorded one legged standing time changed by 5 seconds or more (Hawk and Cambron, 2009).

Table 3.3 Summary of Outcome Measure Requirements for Inclusion and Minimal Clinically Important Change (MCIC)

	Requirements for Inclusion	MCIC
WOMAC	> 500mm, < 1680mm	20%
OTE	No minimum requirement	2.1 points (30%)
BDI	> 21 points	No criteria found in literature
BBS	< 45 points*	6 points
ROM	No minimum requirement	5 -10°
OLST	< 10 seconds*	< 5 seconds

** patients failing to reach these minimum requirements were included, but monitored in an impaired balance subgroup*

3.7 Measurement Frequency

All subjective and objective data was collected at pre-visit one (baseline), pre-visit four and at one week follow-up, with the exception of the McMaster Overall Therapy Effectiveness (OTE) Tool, which was not collected at baseline.

Table 3.4 Summary of Treatment and/or Measurement Frequency

Week	Visit	Group A	Group B	Group C
1	1	**Readings 1** (WOMAC; OLST/BBS; ROM; BDI) MMT	**Readings 1** Rehabilitation	**Readings 1** MMT and Rehabilitation
	2	MMT	Rehabilitation (optional)	MMT and Rehabilitation
2	3	MMT	Rehabilitation (optional)	MMT and Rehabilitation
	4	**Readings 2** (WOMAC; OLST/BBS; ROM; BDI; OTE) MMT	**Readings 2** Rehabilitation	**Readings 2** MMT and Rehabilitation
3	5	MMT	Rehabilitation (optional)	MMT and Rehabilitation
	6	MMT	Rehabilitation (optional)	MMT and Rehabilitation
4	7	**Readings 3** (WOMAC; OLST/BBS; ROM; BDI; OTE)	**Readings 3**	**Readings 3**

3.8 Manual and Manipulative Therapy (MMT) Procedure

3.8.1 Lumbar Spine and Pelvis

MMT was applied where needed to the lumbopelvic spine using diversified techniques, such as high velocity low amplitude (HVLA) manipulation or mobilisation as outlined in Schafer and Faye (1989) and/or Bergmann and Peterson (2002) and / or Byfield (2005).

3.8.2 Hip

The patient was placed in a supine position with the involved limb placed in slight abduction to minimise likelihood of femoral-acetabular impingement occurring at the hip joint. The researcher's encircled the participant's thigh, just above the knee joint, with either their hands or forearms and applied long-axis traction. The first manipulation was applied with the joint in 30° flexion, 30° abduction and slight lateral rotation (maximum loose packed position). Subsequent manipulations were applied with the hip joint in more positions of greater motion restriction, which varied between participants. A maximum of five manipulations were applied at each treatment session, with the final manipulation applied in the most restricted position. In order to assist relaxation, active assisted movements of the hip were performed.

Minimal side-to-side variation in traction and passive hip flexion end feel post-manipulation was considered successful (Hoeksma *et al*., 2004).

3.8.3 Knee

The various manipulative therapies performed on the knee joint are outlined in Table 3.5. Additionally, use of HVLA knee manipulation methods as per Tucker, Brantingham and Myberg (2003) were also be utilised when indicated and tolerated.

Table 3.5 Knee Range of Motion Impairments of the Knee Addressed by Manual Therapy

Impairment	Manual Intervention	Typical Delivery
Loss of extension	Manual mobilisation through range of motion [ROM] and knee extension at end range - Knee extension - Knee extension with abduction - Knee extension with adduction	Mobilisation grades III and IV to III++ and IV++ 2-6 bouts of 30 seconds per manual technique
Loss of flexion	Manual mobilisation through ROM and knee flexion at end range - Knee flexion - Knee flexion with internal rotation	Mobilisation grades III and IV to III+ and IV+ 2-6 bouts of 30 seconds per manual technique
Loss of patellar glide	Mobilisation of patella in 5°-10° of knee flexion - Medial - Lateral - Caudal - Cephalad	Mobilisation grades IV to IV++ 2-6 bouts of 30 seconds per manual technique
Muscle tightness	Manual stretches on length of muscle - Quadriceps femoris - Hamstrings - Gastrocnemius - Iliopsoas - Adductors - Tensor fascia lata (TFL) and Iliotibial band (ITB)	Sustained manual stretch of 12-30 seconds duration repeated 1-3 times per muscle
Soft tissue tightness	Soft tissue mobilisation - Suprapatellar and infrapatellar regions - Medial and lateral joint capsules - Popliteal fossa	Circular fingertip and palm pressure mobilisation at the depth of the capsule of retinaculum for 1-3 bouts of 30 seconds per area

(Adapted from Deyle *et al.,* 2005)

3.8.4 Foot and Ankle

3.8.4.1 Distal Talofibular Joint

Manual therapy was indicated when restriction of anterior to posterior glide was noted on motion palpation. The participant was required to lie in a supine position with the affected limb flexed at the knee and ankle such that their foot was flat on the bed. The researcher stood at the foot of the bed and clasped the ankle with each hand on either malleolus. The technique used varying grades of mobilisation or a thrust in the anterior to posterior direction combined with slight external rotation of the leg (Bergmann and Peterson, 2002; Byfield, 2005).

This restriction was commonly found in individuals with decreased dorsiflexion and post ankle injuries (O'Brien and Vicenzino, 1998).

3.8.4.2 Tibiotalar Joint

Green *et al*. (2001) and Whitman *et al.* (2009) indicated an oscillating mobilisation (Grade 3 to 4), with or as a HVLA thrust when appropriate, for decreased ankle dorsiflexion or long axis distraction. It was performed with the participant in a supine position with the knee extended and the affected ankle supported at the foot of the bed. The researcher stood at the ipsilateral side of the bed and supported the foot by cupping the calcaneus with the caudad hand. The cephalad hand grasped the anterior aspect of the ankle, above the level of the malleoli, with a web-type contact. A force was directed in an anterior to posterior vector (Bergmann and Peterson, 2002; Byfield, 2005).

3.8.4.3 General Midtarsal Joint

Manual therapy was indicated when a side-to-side difference in general ROM of the midfoot was noted. The participant was required to lie in a supine position with the researcher positioned at the foot of the bed. The researcher contacts the midfoot with one hand and stabilises the proximal foot, by cupping the calcaneus, with the other hand. General foot mobility is determined by circumduction of the midfoot in a clockwise direction, followed by an anti-clockwise direction, such that a figure-of-8 mobilisation pattern is created (Bergmann and Peterson, 2002; Byfield, 2005).

3.8.4.4 Tarsal Joint Plantar To Dorsal

This technique is used when there is a side-to-side difference in plantar to dorsal mobility on motion palpation of the cuboid bone (or other small tarsal). It is performed by having the patient lie prone on a bed with the researcher standing at the foot of the bed. A double thumb contact is taken on the plantar aspect of the affected segment, with the knee and ankle in slight flexion. A rapid plantar to dorsal thrust is applied to the foot, causing extension of the knee and ankle, resulting in a whip-like motion (Bergmann and Peterson, 2002; Byfield, 2005).

3.8.4.5 First Metatarsophylangeal Joint and Sesamoid Bones

Indications of MMT of the first ray were considered as loss of passive ROM and springy end feel in the dorsiflexion (65-75°) and plantarflexion (45°) directions. The participant was required to assume a supine position. The researcher's caudad hand contacted the distal

phalanx of the hallux, while the cephalad hand contacted the forefoot, just proximal to the metatarsals. Oscillation in axial elongation was applied using Grade 1-4 mobilisation.

Depending on participant's tolerance, HVLA axial elongation of the first MTPJ was applied by positioning the patient in the supine position, with the ipsilateral calcaneus rested anterior to the researcher's bended knee, in order to stabilise the foot. The researcher's distal thumbs crossed and grasped the proximal phalanx of a slightly plantarflexed hallux. Slight traction was applied until resistance was felt, followed by a Grade 5 manipulative thrust in the proximal to distal direction (Bergmann and Peterson, 2002; Byfield, 2005).

Indications for sesamoid mobilisation included decreased dorsiflexion and increased pain in the first MTPJ. The patient was to lie supine while the researcher contacted the proximal sesamoids with the thumb of their cephalad hand. The researcher's caudad hand stabilised the hallux and distal foot while Grade 3 and 4 mobilisation was applied in a proximal to distal direction (Bergmann and Peterson, 2002; Byfield, 2005).

3.8.4.6 Second to Fifth Metatarsophylangeal and Intermetatarsal Joint

Axial elongation of the second to fifth MTP joint and interphylangeal joints was indicated if there was side-to-side difference in ROM and joint play. The participant was placed in the supine position and the researcher contacted the distal pad of the toe with their thumb and PIPJ of the index finger. The contact fingers were reinforced with the pisiform of the other hand and an axial elongation mobilisation or thrust was applied.

All MMT of the toes was contra-indicated in the presence of rheumatoid arthritis, gout and diabetes mellitus (Bergmann and Peterson, 2002; Byfield, 2005).

3.9 Rehabilitation Procedure

The same rehabilitation protocol, comprising strengthening exercises, stretches and ROM exercises, was prescribed to all patients in Group 2 and Group 3. The various aspects, as well as exercise prescription, are described in the Table 3.6, Table 3.7 and Table 3.8.

Table 3.6 Strengthening Exercises for Rehabilitation Protocol

Exercise	Performance	Repetition
Static quad sets in knee extension	- Perform daily - Patient is positioned fully supine or supine supported on elbows with the knee in full extension - Patient contracts the quadriceps femoris muscle and pushes the knee down while maintaining the foot in full dorsiflexion	- Hold each contraction for 6 seconds with a 10 second rest between repetitions - Repeat x 10
Standing terminal knee extension	- Perform 3 x per week - Patient stands with a resistive band or a cuff from a weighted pulley machine behind a slightly flexed knee - Patient contracts the gluteal and quadriceps femoris muscles to fully straighten the hip and knee	- Hold each contraction for 3 seconds - Repeat x 10 - Increase resistance as tolerated
Closed-chain progression:	- Patient should progress to the most challenging activity that he/she can successfully complete with minimal or no pain	- Patient performs one of the following activities 3 x per week
1. Seated leg press	- Patient is seated holding a resistive band in both hands - Patient places his/her foot against the band, then straightens the knee by contracting the gluteal and quadriceps femoris muscles	- Hold each contraction 3 seconds with knee as straight as possible - Slowly return to starting position and repeat x 10 - Progress to bands of increasing resistance and additional bouts
2. Partial squats weight-lessened with arm support as needed	- Patient stands with arm support as needed - Patient performs a partial squat, keeping the knees centred over the feet - Return to standing by contracting the quadriceps femoris and gluteal muscles	- Hold each contraction 3 seconds with hips and knees as straight - Repeat x10 - Progress to full body weight without support and additional bouts
3. Step-ups	- Patient stands in front of a low step - Patient places foot of involved leg on step and brings body over foot to stand on the step - Use as little push-off assistance from the contralateral foot as possible - Step down with contralateral foot	- Slow repeat for 30 seconds - Progress to increased height of the step and additional bouts - Alternate legs if both knees are involved

(Adapted from Deyle *et al.*, 2005)

Table 3.7 Stretch Exercises for Rehabilitation Protocol

Exercise	Performance	Repetition
Standing calf stretch	- Perform daily - Patient stands with the heel of the foot on the ground behind the patient; the toes point straight ahead - The patient leans forward until a moderate pull is perceived in the calf musculature - The patient may use his or her arms for support against a wall or furniture as needed	Hold for 30 seconds and repeat x 3
Supine hamstring muscle stretch	- Perform daily - The patient is positioned supine with the contralateral lower extremity maintained as straight as possible - The ipsilateral hip is flexed to 90° - The knee is straightened and the proximal lower leg supported with the hands until a moderate pull is perceived in the anterior thigh and calf - Ipsilateral ankle should be dorsiflexed	Hold for 30 seconds and repeat x 3

Table 3.7 Stretch Exercises for Rehabilitation Protocol continued…

Prone quadriceps femoris muscle stretch	- Perform daily - Patient is positioned prone with both hips and knees extended - A strap is placed around the ipsilateral ankle and brought posteriorly and superiorly over the ipsilateral shoulder - The patient grasps the strap in the ipsilateral hand and bends the knee by straightening his/her elbow and pulling on the strap - The knee is progressively flexed until a gentle stretch in perceived in the anterior thigh	Hold for 30 seconds and repeat x 3

(Adapted from Deyle *et al.,* 2005)

Table 3.8 Range of Motion Exercises for Rehabilitation Exercises

Exercise	Performance	Repetitions
Knee in mid-extension to full extension	- Perform once daily - Patient is positioned supine or supine supported on elbows - Knee is brought to 45° of flexion with the ipsilateral foot sliding on the surface that the patient is lying on - The knee is then fully extended with a strong quadriceps femoris muscle contraction against any limitation to full knee extension	Two 30 second bouts with 3 second hold at end range
Knee in mid-flexion to full flexion	- Perform once daily - Patient is positioned supine or supine supported on elbows - Knee id brought to full flexion with assistance of the upper extremity or a strap - A gentle challenge to end-range flexion is sustained	Two 30 second bouts with 3 second hold at end range
Stationary bicycle	- Perform once daily - Knees should be at a nearly full extension at bottom of pedal stroke	5 minutes, increasing time as tolerated

(Adapted from Deyle *et al.,* 2005)

3.10 Statistical Methodology

IBM SPSS 19 was used to analyse the data. A *p*-value <0.05 was considered as statistically significant. Repeated measures ANOVA testing was used to examine the effect of time (using the Wilks' lambda statistic) in intra-group analyses. In order to compare treatment effects over time between the three treatments, inter-group analyses used repeated measures ANOVA testing, and the time*group interaction effect was examined (again using the Wilks' lambda statistic). Profile plots were generated in order to examine the direction and trend of the effect. Kruskal-Wallis tests were used to compare median OTE 2 score between the three treatment groups at each follow-up time point (Esterhuizen, 2012).

If any patients “drop out” of the study their data collected was kept for intent to treat analysis. Also, the reason for dropping out was noted and commented thereon, especially if it is due to adverse side effects (Esterhuizen, 2012).

- Intent to treat analysis was done to avoid the effect of drop out, reducing any biases to the statistical analysis. Therefore, intention to treat analysis provided information about the initial treatment intent, not the treatment eventually administered.
- Any drop outs prior to visit 4 were replaced with new patients to ensure that at least 60 patients complete the minimum treatment process. However, everyone who began the treatment was considered to be part of the clinical trial, whether they completed or not.

Full application of intention to treat can only be performed where there is complete outcome data for all randomised patients. Therefore, all patients who dropped out of the study, or were excluded, were asked to return so that where possible final outcome measurements could be made (Esterhuizen, 2012).

CHAPTER FOUR

RESULTS

4.1 Introduction

This chapter contains the statistical analysis of subjective and objective data acquired from patients over the duration of the four week assessment period.

Subjective data obtained was in the form of the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC), the McMaster Overall Therapy Effectiveness (OTE) Tool, the Beck Depression Inventory (BDI) and the Berg Balance Scale (BBS). The WOMAC scores were further described by the overall WOMAC score and by its three subcategories; pain (WOMAC-P), stiffness (WOMAC-S), and disability (WOMAC-D). Likewise the OTE is described by its two subdivisions, Overall Treatment Evaluation (OTE.1) and Overall Treatment Effect (OTE.2).

Objective data was recorded by means of the One Leg Standing test (OLST), and inclinometer measures to determine Range of Motion in flexion (ROMf) and extension (ROMe) at the knee joint.

The following abbreviations have been used in this chapter:

df. or diff.	Difference
F	Frequency
p or Sig.	Level of significance
%	Percentage
n	Sample size/ Number
SD	Standard deviation

The following units were used in the tables:

Age	Years (yrs)
Duration	Months (mo)
Height	Centimetres (cm)
OLST	Seconds (sec)
ROMf and ROMe	Degrees (°)
Weight	Kilograms (kg)

4.2 The Data

There were two forms of data collected in this research, primary and secondary data.

4.2.1 Primary Data

There were six types of primary data:

1. Patient's response to the Western Ontario and McMaster Universities Osteoarthritis Index (Appendix J).
2. Patient's response to the Beck Depression Inventory (Appendix L).
3. Patient's response to the Berg Balance Scale (Appendix M).
4. Patient's response to the McMaster Overall Therapy Effectiveness Tool (Appendix K).
5. Patient's response to the One Leg Standing Test.
6. Patient's readings on Inclinometer (Appendix N) measurement of flexion and extension range of motion at the knee joint.

4.2.2 Secondary Data

This was in the form of published documentation and accepted theories on osteoarthritis of the knee, rehabilitation, and manipulative therapies.

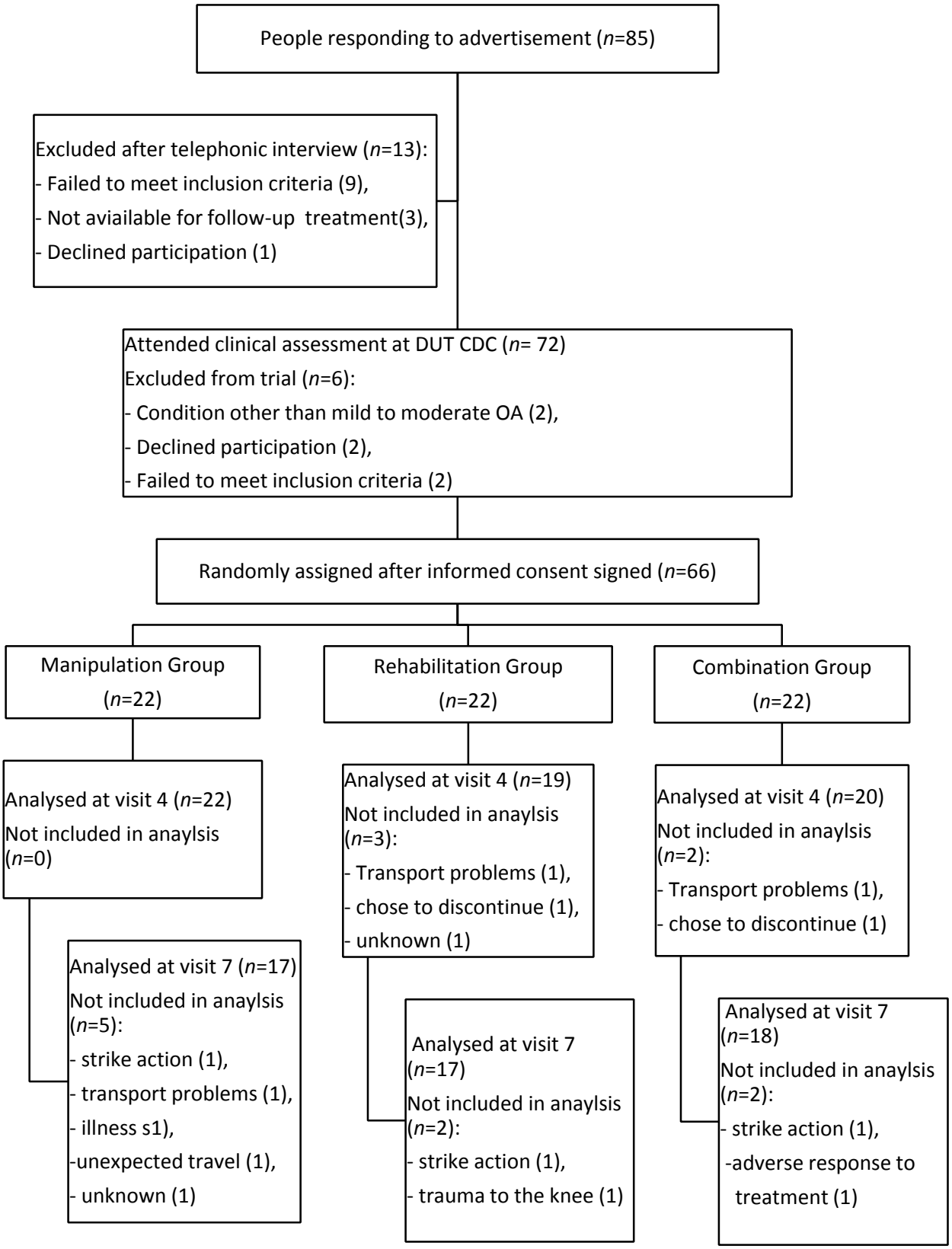

Figure 4.1 Modified Consolidated Standard for Reporting Clinical Trials (CONSORT) diagram

(Schultz, Altman and Moher, 2010)

4.3 Physical Characteristics and Demographic Data

The physical characteristics of the patients (n= 61) who completed this trial are summarised in Table 4.1.1 and Table 4.1.2. The sample was made up of patients between the age of 38 and 80, who had suffered from osteoarthritis of the knee for longer than a year.

There were no statistically significant differences between the three treatment groups in terms of any of the below demographic or clinical factors.

Table 4.1.1 Distribution of Sample According to Demographic Characteristics (Qualitative)

				Group					
				Manipulation		Rehabilitation		Combination	
		n	%	n	%	n	%	n	%
Gender	Female	40	65.6%	16	72.7%	12	63.2%	12	60.0%
	Male	21	34.4%	6	27.3%	7	36.8%	8	40.0%
Occupation	Domestic worker	2	3.3%	1	4.5%	0	0.0%	1	5.0%
	Housewife	14	23.0%	7	31.8%	5	26.3%	2	10.0%
	Pensioner	24	39.3%	7	31.8%	7	36.8%	10	50.0%
	Retired	3	4.9%	1	4.5%	2	10.5%	0	0.0%
	Shop worker/ owner	3	4.9%	1	4.5%	1	5.3%	1	5.0%
	Teacher	3	4.9%	1	4.5%	0	0.0%	2	10.0%
	Unemployed	4	6.6%	0	0.0%	2	10.5%	2	10.0%
	Other	8	13.1%	4	18.2%	2	10.5%	2	10.0%
Onset	Insidious	52	85.2%	19	86.4%	18	94.7%	15	75.0%
	Trauma	9	14.8%	3	13.6%	1	5.3%	5	25.0%
Side	Left	20	45.9%	7	31.8%	7	36.8%	6	30.0%
	Right	34	55.7%	13	59.1%	9	47.4%	12	60.0%
	Both	7	11.5%	2	9.1%	3	15.8%	2	10.0%

Table 4.1.2 Distribution of Sample According to Demographic Characteristics (Quantitative)

	Group					
	Manipulation		Rehabilitation		Combination	
	Mean	SD	Mean	SD	Mean	SD
Age (yrs)	63.1	10.8	62.1	10.8	62.1	10.3
Duration (mo)	84.8	82.0	55.3	59.1	103.6	145.3
Height (cm)	166.8	9.2	167.4	17.0	164.4	8.6
Weight (kg)	80.8	15.1	85.5	17.4	85.9	22.1

4.4 Baseline Readings

This was done in order to establish that there were no pre-existing differences between the groups which could have influenced the outcomes after treatment. In terms of baseline outcome values, only WOMAC Stiffness (WOMAC-S) showed a statistically significant difference between treatment groups at baseline (p=0.040) (Table 4.2.2). Bonferroni post hoc tests showed that rehabilitation and combination groups differed significantly (p=0.043) (Table 4.2.3) while none of the other pairs differed.

Table 4.2.1 Comparison of Baseline Outcomes between Groups

	Group					
	Manipulation		Rehabilitation		Combination	
	Mean	SD	Mean	SD	Mean	SD
WOMAC Overall	1 076.1	336.0	960.6	277.6	1,145.9	330.1
WOMAC-P	221.9	82.9	184.5	67.5	206.1	87.2
WOMAC-S	100.0	44.3	90.0	38.6	124.6	44.7
WOMAC-D	753.8	245.1	687.1	222.1	815.3	256.8
BDI	9.6	5.8	8.8	6.4	8.7	6.7
OLST	13.4	7.7	14.7	7.7	12.5	6.9
BBS	42.0	5.6	42.6	6.7	43.0	7.3
ROMf	93.2	18.5	99.7	20.4	94.2	15.8
ROMe	6.0	1.9	5.9	1.7	5.1	1.7

Table 4.2.2
ANOVA Comparison of Mean Baseline Outcomes between the Three Treatment Groups

		Sum of Squares	df	Mean Square	F	Sig.
WOMAC Overall	Between Groups	340 185.422	2	170 092.711	1.693	0.193
	Within Groups	5 828 061.562	58	100 483.820		
	Total	6 168 246.984	60			
WOMAC-P	Between Groups	14 276.233	2	7 138.116	1.116	0.334
	Within Groups	370 893.505	58	6 394.716		
	Total	385 169.738	60			
WOMAC-S	Between Groups	12 478.686	2	6 239.343	3.414	0.040
	Within Groups	105 999.905	58	1 827.585		
	Total	118 478.590	60			
WOMAC-D	Between Groups	160 012.958	2	80 006.479	1.364	0.264
	Within Groups	3 402 969.403	58	58 671.886		
	Total	3 562 982.361	60			

Table 4.2.2 continued.
ANOVA Comparison of Mean Baseline Outcomes between the Three Treatment Groups …

		Sum of Squares	df	Mean Square	F	Sig.
BDI	Between Groups	11.570	2	5.785	0.147	0.864
	Within Groups	2 286.167	58	39.417		
	Total	2 297.738	60			
OLST	Between Groups	48.872	2	24.436	0.441	0.645
	Within Groups	3 212.373	58	55.386		
	Total	3 261.246	60			
BBS	Between Groups	4.539	2	2.270	0.053	0.948
	Within Groups	855.200	20	42.760		
	Total	859.739	22*			
ROMf	Between Groups	484.570	2	242.285	0.723	0.489
	Within Groups	19 428.578	58	334.975		
	Total	19 913.148	60			
ROMe	Between Groups	9.395	2	4.697	1.486	0.235
	Within Groups	183.294	58	3.160		
	Total	192.689	60			

* Only those participants who failed the OLST went on to complete the BBS

Table 4.2.3
Bonferroni Post Hoc Comparison of Mean WOMAC Stiffness Scores between the Three Treatment Groups

(I) Group	(J) Group	Mean Difference (I-J)	Standard Error	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Manipulation	Rehabilitation	9.955	13.389	1.000	-23.05	42.96
	Combination	-24.595	13.208	0.203	-57.16	7.97
Rehabilitation	Manipulation	-9.955	13.389	1.000	-42.96	23.05
	Combination	-34.550(*)	13.696	0.043	-68.31	-0.79
Combination	Manipulation	24.595	13.208	0.203	-7.97	57.16
	Rehabilitation	34.550(*)	13.696	0.043	0.79	68.31

* The mean difference is significant at the 0.05 level.

4.5 Assessment of the Treatment Effect

4.5.1 Intra-group Analysis

4.5.1.1 Subjective Outcomes Findings

Subjective data obtained was in the form of the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC), the Beck Depression Inventory (BDI), the Berg Balance Scale (BBS) and the McMaster Overall Therapy Effectiveness (OTE) Tool.

4.5.1.1.1 Western Ontario and McMaster Universities Osteoarthritis Index

WOMAC Overall Score

In each of the three treatment groups, there was a statistically significant decrease in the mean value for the overall WOMAC score over time. For the purposes of this study, successful clinical change was defined as a minimum of 20% change (Barr *et al*., 1994). Therefore, all groups achieved statistical and clinical improvements, with the Manipulation and Combination groups being significant at the 99% confidence interval and the rehabilitation group at the 95% confidence interval.

Table 4.3.1
Means and Standard Deviations in Overall WOMAC Scores at Baseline, Visit 4 and Visit 7

	Group					
	Manipulation		Rehabilitation		Combination	
	Mean	SD	Mean	SD	Mean	SD
Baseline	1 076.1	336.0	960.6	277.6	1,145.9	330.1
Visit 4	771.1	376.2	709.2	229.1	736.4	384.3
Visit 7	598.6	404.4	638.3	234.7	567.4	436.2

Table 4.3.2
Mean Difference and Percentage Change in Overall WOMAC Scores from Baseline to Visit 4 and Visit 7

	Group					
	Manipulation		Rehabilitation		Combination	
	Mean diff.	Change (%)	Mean diff.	Change (%)	Mean diff.	Change (%)
Baseline – Visit 4	305.0 (±40.2)	28.3%	251.4 (±48.5)	26.2%	409.5 (±54.2)	35.7%
Visit 4 – Visit 7	172.5 (±28.2)	22.4%	70.9 (±5.6)	10.0%	169.0 (±51.9)	22.9%
Baseline – Visit 7	447.5 (±68.4)	44.4%	322.3 (±42.9)	33.6%	578.5 (±106.1)	50.5%

Table 4.3.3
Intra-Group Comparison of the Effect on Overall WOMAC Scores of Each Intervention Group

Multivariate Tests[b]							
Group	Effect		Value	F	Hypothesis df	Error df	Sig.
Manipulation	time						
		Wilks' Lambda	0.302	23.121[a]	2.000	20.000	<0.001
Rehabilitation	time						
		Wilks' Lambda	0.511	8.147[a]	2.000	17.000	0.003
Combination	time						
		Wilks' Lambda	0.228	30.456[a]	2.000	18.000	<0.001

a. Exact statistic
b. Design: Intercept
Within Subjects Design: time

WOMAC Pain Score

In each of the three treatment groups, there was a statistically significant decrease in mean for the WOMAC Pain score over time. This followed similar trends to the WOMAC overall score in terms of the confidence intervals within which the results fell.

Table 4.3.4
Means and Standard Deviations in WOMAC Pain Scores at Baseline, Visit 4 and Visit 7

	Group					
	Manipulation		Rehabilitation		Combination	
	Mean	SD	Mean	SD	Mean	SD
Baseline	221.9	82.9	184.5	67.5	206.1	87.2
Visit 4	144.6	104.1	111.3	48.5	136.8	76.2
Visit 7	117.0	109.8	121.2	54.6	111.2	94.8

Table 4.3.5
Mean Difference and Percentage Change in WOMAC Pain Scores from Baseline to Visit 4 and Visit 7

	Group					
	Manipulation		Rehabilitation		Combination	
	Mean diff.	Change (%)	Mean diff.	Change (%)	Mean diff.	Change (%)
Baseline – Visit 4	77.3 (±21.2)	34.8%	73.2 (±19.0)	39.7%	69.3 (±11.0)	33.6%
Visit 4 – Visit 7	27.6 (±5.7)	19.1%	-9.9 (±6.1)	-8.9%	25.6 (±18.6)	18.7%
Baseline - Visit 7	104.9 (±26.9)	47.3%	63.3 (±12.9)	34.3%	94.9 (±7.6)	46.0%

Table 4.3.6
Intra-Group Comparison of the Effect on WOMAC Pain Scores of Each Intervention Group

Multivariate Tests[b]							
Group	Effect		Value	F	Hypothesis df	Error df	Sig.
Manipulation	time						
		Wilks' Lambda	0.451	12.160[a]	2.000	20.000	<0.001
Rehabilitation	time						
		Wilks' Lambda	0.513	8.075[a]	2.000	17.000	0.003
Combination	time						
		Wilks' Lambda	0.418	12.543[a]	2.000	18.000	<0.001

a. Exact statistic
b. Design: Intercept
Within Subjects Design: time

WOMAC Stiffness Score

In each of the three treatment groups, there was a statistically significant decrease in the mean value for the WOMAC Stiffness score over time, which is not dissimilar to the WOMAC Pain score.

Table 4.3.7
Means and Standard Deviations in WOMAC Stiffness Scores at Baseline, Visit 4 and Visit 7

	Group					
	Manipulation		Rehabilitation		Combination	
	Mean	SD	Mean	SD	Mean	SD
Baseline	100.0	44.3	90.0	38.6	124.6	44.7
Visit 4	67.0	44.0	65.7	28.1	76.6	38.1
Visit 7	50.9	39.7	53.4	28.3	53.8	41.4

Table 4.3.8
Mean Difference and Percentage Change in WOMAC Stiffness Scores from Baseline to Visit 4 And Visit 7

	Group					
	Manipulation		Rehabilitation		Combination	
	Mean diff.	Change (%)	Mean diff.	Change (%)	Mean diff.	Change (%)
Baseline – Visit 4	33.0 (±0.3)	33.0%	24.3 (±10.5)	39.7%	48.0(±6.6)	33.6%
Visit 4 – Visit 7	16.1 (±4.3)	24.0%	12.3 (±0.2)	-8.9%	22.8 (±3.3)	18.7%
Baseline - Visit 7	49.1 (±4.6)	49.1%	36.6 (±10.3)	34.3%	70.8 (±3.3)	46.0%

Table 4.3.9
Intra-Group Comparison of the Effect on WOMAC Stiffness Scores of Each Intervention Group

Multivariate Tests[b]							
Group	Effect		Value	F	Hypothesis df	Error df	Sig.
Manipulation	time						
		Wilks' Lambda	0.449	12.264[a]	2.000	20.000	<0.001
Rehabilitation	time						
		Wilks' Lambda	0.509	8.190[a]	2.000	17.000	0.003
Combination	time						
		Wilks' Lambda	0.203	35.298[a]	2.000	18.000	<0.001

a. Exact statistic
b. Design: Intercept
Within Subjects Design: time

WOMAC Disability Score

In each of the three treatment groups, there was a statistically significant decrease in the mean value for the WOMAC Disability score over time.

Table 4.3.10
Means and Standard Deviations in WOMAC Disability Scores at Baseline, Visit 4 and Visit 7

	Group					
	Manipulation		Rehabilitation		Combination	
	Mean	SD	Mean	SD	Mean	SD
Baseline	753.8	245.1	687.1	222.1	815.3	256.8
Visit 4	558.1	266.6	503.5	188.7	523.1	298.0
Visit 7	429.5	280.4	456.3	192.1	401.4	315.5

Table 4.3.11
Mean Difference and Percentage Change in WOMAC Disability Scores from Baseline to Visit 4 and Visit 7

	Group					
	Manipulation		Rehabilitation		Combination	
	Mean diff.	Change (%)	Mean diff.	Change (%)	Mean diff.	Change (%)
Baseline – Visit 4	195.7 (±21.5)	26.0	183.6 (±33.4)	2.67	292.2 (±41.2)	35.8
Visit 4 – Visit 7	128.6 (±13.8)	23.0	47.2 (±3.4)	9.4	121.7 (±17.5)	14.9
Baseline - Visit 7	324.3 (±35.3)	43.0	230.8 (±30.0)	33.6	413.9 (±58.7)	50.8

Table 4.3.12
Intra-Group Comparison of the Effect on WOMAC Disability Scores of Each Intervention Group

Multivariate Tests[b]							
Group	Effect		Value	F	Hypothesis df	Error df	Sig.
Manipulation	Time						
		Wilks' Lambda	0.325	20.766[a]	2.000	20.000	<0.001
Rehabilitation	Time						
		Wilks' Lambda	0.516	7.978[a]	2.000	17.000	0.004
Combination	Time						
		Wilks' Lambda	0.239	28.696[a]	2.000	18.000	<0.001

a. Exact statistic
b. Design: Intercept
Within Subjects Design: time

WOMAC Summary of the percentage improvements over time

From Table 4.3.13, it can be seen that overall changes (visit 1 to visit 4; visit 4 to visit 7 and visit 1 to visit 7) within the WOMAC seem to favour the combination groups, followed by the manipulation group and lastly, the rehabilitation only group.

Table 4.3.13 Summary of Percentage Change in WOMAC Scores between Treatment Groups

		Manipulation	Rehabilitation	Combination
WOMAC Overall	V1-v4 V4-v7 V1-v7	28.3 22.4 44.2	26.2 10.0 33.6	35.7 22.9 50.5
WOMAC-P	V1-v4 V4-v7 V1-v7	34.8 19.1 47.3	39.7 -8.9 34.3	33.6 25.6 46.0
WOMAC-S	V1-v4 V4-v7 V1-v7	33.0 24.0 49.1	24.3 18.7 40.7	38.5 29.8 56.8
WOMAC -D	V1-v4 V4-v7 V1-v7	26.0 23.0 43.0	26.7 9.4 33.6	35.8 14.9 50.8

4.5.1.1.2 Beck Depression Inventory

Only in the combination group was there a decrease in BDI score, which was statistically significant (p=0.006) over time.

Table 4.3.14
Means, Standard Deviations and Change in BDI Scores at Baseline, Visit 4 and Visit 7

	Group								
	Manipulation			Rehabilitation			Combination		
	Mean	SD	Change	Mean	SD	Change	Mean	SD	Change
Baseline	9.6	5.8		8.8	6.4		8.7	6.7	
Visit 4	8.5	4.9	1.1 (±0.9)	8.1	6.4	0.7 (±0.0)	7.2	5.2	1.5 (±1.5)
Visit 7	7.2	5.1	1.3 (±0.2)*	7.8	5.7	0.3 (±0.7)*	6.4	4.7	0.8 (±0.5)*
			2.4 (±0.7)**			1.0 (±0.7)**			2.3 (±2.0)**

* Change between visit 4 and visit 7
** Total change from baseline to visit 7

Table 4.3.15 Intra-Group Comparison of the Effect on BDI Scores of Each Intervention Group

Multivariate Tests[b]							
Group	Effect		Value	F	Hypothesis df	Error df	Sig.
Manipulation	time						
		Wilks' Lambda	0.791	2.637[a]	2.000	20.000	0.096
Rehabilitation	time						
		Wilks' Lambda	0.752	2.798[a]	2.000	17.000	0.089
Combination	time						
		Wilks' Lambda	0.568	6.856[a]	2.000	18.000	0.006
a. Exact statistic b. Design: Intercept Within Subjects Design: time							

4.5.1.1.3 Berg Balance Scale

There was a statistically significant change over time for BBS values in each of the three groups (all at the 95% confidence interval). Score of 45 or lower representing increased risk of fall (Lajoie and Gallagher, 2004), with a 6 point change being determined as clinically significant (Stevenson, 2001). Therefore, although the change in BBS was statistically significant and the mean visit 7 scores no longer represented an increased risk of fall, the change in BBS scores was not deemed clinically significant.

Table 4.3.16
Means, Standard Deviations and Change in BBS Scores at Baseline, Visit 4 and Visit 7

	Group								
	Manipulation			Rehabilitation			Combination		
	Mean	SD	Change	Mean	SD	Change	Mean	SD	Change
Baseline	42.0	5.6		42.6	6.7		43.0	7.3	
Visit 4	45.9	4.5	3.9 (±1.1)	44.4	6.3	1.8 (±0.4)	45.4	4.6	2.4 (±2.7)
Visit 7	47.0	3.9	1.1 (±0.6)*	45.6	6.5	1.2 (±0.2)*	46.5	4.2	1.1 (±0.4)*
			5.0 (±1.7)**			3.0 (±0.2)**			3.5 (±3.1)**

* Change between visit 4 and visit 7
** Total change from baseline to visit 7

Table 4.3.17 Intra-Group Comparison of the Effect on BBS Scores of Each Intervention Group

Multivariate Tests[b]							
Group	Effect		Value	F	Hypothesis df	Error df	Sig.
Manipulation	time						
		Wilks' Lambda	0.326	6.192[a]	2.000	6.000	0.035
Rehabilitation	time						
		Wilks' Lambda	0.103	13.000[a]	2.000	3.000	0.033
Combination	time						
		Wilks' Lambda	0.345	5.694[a]	2.000	6.000	0.041

a. Exact statistic
b. Design: Intercept
Within Subjects Design: time

4.5.1.1.4 McMaster Overall Therapy Effectiveness Tool

Overall Treatment Evaluation (OTE.1)

Only the combination group showed a statistically significant change over time (p=0.045), however, this change was not considered clinically significant as it was not greater than a 30% or 2.1 point improvement.

Table 4.3.18
Means, Standard Deviations and Change in Overall Treatment Evaluation at Visit 4 and Visit 7

	Group								
	Manipulation			Rehabilitation			Combination		
	Mean	SD	Change	Mean	SD	Change	Mean	SD	Change
Visit 4	4.0	1.4		3.4	1.4		3.9	2.8	
Visit 7	5.4	1.1	1.4 (±0.3)	4.4	1.3	1.0 (±0.1)	5.6	1.4	1.7 (±1.4)

Table 4.3.19 Intra-Group Comparison of the Effect on OTE.1 Scores of Each Intervention Group

Multivariate Tests[b]							
Group	Effect		Value	F	Hypothesis df	Error df	Sig.
Manipulation	time						
		Wilks' Lambda	0.888	2.646[a]	1.000	21.000	0.119
Rehabilitation	time						
		Wilks' Lambda	0.993	0.124[a]	1.000	18.000	0.728
Combination	time						
		Wilks' Lambda	0.805	4.607[a]	1.000	19.000	0.045

a. Exact statistic
b. Design: Intercept
Within Subjects Design: time

Table 4.3.20 Dichromatisation of Overall Treatment Evaluation at Visit 4 and Visit 7

	Group								
	Manipulation			Rehabilitation			Combination		
	Better	The same	Worse	Better	The same	Worse	Better	The same	Worse
Visit 4	21	1	0	18	1	0	18	1	1
Visit 7	22	0	0	16	2	1	19	0	1

Overall Treatment Effect (OTE.2)

There were no statistically significant differences within any of the groups between visit 4 and visit 7 measurements for the OTE.2 scale at the 0.05 level of significance. In the rehabilitation group, the difference was almost statistically significant (p=0.058). The median score in that group increased from 4 to 5 from visit 4 to visit 7. In the other two groups, there was no change in median score from visit 4 to visit 7.

Table 4.3.21
Means, Standard Deviations and Change in Overall Treatment Effect at Visit 4 and Visit 7

	Group								
	Manipulation			Rehabilitation			Combination		
	Mean	SD	Change	Mean	SD	Change	Mean	SD	Change
Visit 4	4.4	1.6		3.6	2.5		4.5	2.7	
Visit 7	5.3	1.3	0.9 (±0.3)	4.9	1.5	1.3 (±1.0)	5.6	1.2	1.1 (±1.5)

Table 4.3.22 Intra-Group Comparison of the Effect on OTE.2 Scores of Each Intervention Group

Group	Null Hypothesis	Test	Sig.	Decision
Manipulation	The median of differences between OTE.2 at visit 4 and visit 7 equals 0.	Related-Samples Wilcoxon Signed Rank Test	0.785	Retain the null hypothesis
Rehabilitation			0.058	Retain the null hypothesis
Combination			1.000	Retain the null hypothesis

4.5.1.2 Objective Outcome Findings

4.5.1.2.1 One Leg Standing Test

All three groups showed statistically significant change over time for OLST, with the greatest improvement, 4.4 (±0.4) seconds, and statistical significance (p<0.001) in the combination group (viz. the only group to achieve the 99% confidence interval).

Table 4.4.1
Means, Standard Deviations and Change in OLST Scores at Baseline, Visit 4 and Visit 7

	Group								
	Manipulation			Rehabilitation			Combination		
	Mean	SD	Change	Mean	SD	Change	Mean	SD	Change
Baseline[a]	13.4	7.7		14.7	7.7		12.5	6.9	
Subgroup[b]	5.6 (*n*= 8)	2.3		4.2 (*n*= 5)	4.1		7.2 (*n*= 10)	6.0	
Visit 4	7.0	3.2	1.4 (±0.9)	7.0	5.1	2.8	8.6	6.0	1.4 (±0.0)
Visit 7	7.6	3.0	0.6 (±0.2)*	8.2	4.2	1.2 (±1.0)*	11.6	6.4	3.0 (±0.4)*
			2.0 (±0.7)**			4.0 (±0.1)**			4.4 (±0.4)**

[a] Whole sample (*n* = 61)
[b] Only those who failed at initial (*n* = 23)
* Change between visit 4 and visit 7
** Total change from baseline to visit 7

Table 4.4.2 Intra-Group Comparison of the Effect on OLST Scores of Each Intervention Group

Multivariate Tests[b]							
Group	Effect		Value	F	Hypothesis df	Error df	Sig.
Manipulation	time						
		Wilks' Lambda	0.275	7.902[a]	2.000	6.000	0.021
Rehabilitation	time						
		Wilks' Lambda	0.090	15.254[a]	2.000	3.000	0.027
Combination	time						
		Wilks' Lambda	0.124	28.279[a]	2.000	8.000	<0.001

a. Exact statistic
b. Design: Intercept
Within Subjects Design: time

4.5.1.2.2 Range of Motion

Flexion (ROMf) [Normal range = 130-150°]

In each of the three treatment groups, there was a statistically significant increase in the mean value for the ROMf over time (only the rehabilitation group fell within the 95% confidence interval, with the other two groups falling into the 99% confidence interval). A clinically significant increase in ROM is indicated by 5-10° of improved movement (Evans, 2001), which was obtained by all treatment groups at visit 4 and visit 7.

Table 4.4.3
Means, Standard Deviations and Change in ROMf Readings at Baseline, Visit 4 and Visit 7

	Group								
	Manipulation			Rehabilitation			Combination		
	Mean	SD	Change	Mean	SD	Change	Mean	SD	Change
Baseline	93.2	18.5		99.7	20.4		94.2	15.8	
Visit 4	103.6	17.2	10.4 (±1.3)	107.5	15.9	7.8 (±4.5)	101.8	14.9	7.6 (±0.9)
Visit 7	106.5	18.8	2.9 (±1.6)*	108.6	16.5	1.1 (±0.6)*	105.6	14.4	3.8 (±0.5)*
			13.3 (±0.3)**			8.9 (±3.9)**			11.4 (±1.4)**

* Change between visit 4 and visit 7
** Total change from baseline to visit 7

Table 4.4.4 Intra-Group Comparison of the Effect on ROMf Scores of Each Intervention Group

Multivariate Tests[b]							
Group	Effect		Value	F	Hypothesis df	Error df	Sig.
Manipulation	time						
		Wilks' Lambda	0.121	72.870[a]	2.000	20.000	<0.001
Rehabilitation	time						
		Wilks' Lambda	0.509	8.205[a]	2.000	17.000	0.003
Combination	time						
		Wilks' Lambda	0.359	16.078[a]	2.000	18.000	<0.001

a. Exact statistic
b. Design: Intercept
Within Subjects Design: time

Extension (ROMe) [Normal range = 0°, or 10-15° hyperextension]

In each of the three treatment groups, there was a statistically significant increase in the mean value for the ROMe over time. However, no group achieved a clinically significant change of 5-10° (Evans, 2001).

Table 4.4.5 Intra-Group Comparison of the Effect on ROMe Scores of Each Intervention Group

	Group								
	Manipulation			Rehabilitation			Combination		
	Mean	SD	Change	Mean	SD	Change	Mean	SD	Change
Baseline	6.0	1.9		5.9	1.7		5.1	1.7	
Visit 4	7.3	2.0	1.3 (±0.1)	6.6	1.5	0.7 (±0.2)	5.7	1.4	0.6 (±0.3)
Visit 7	7.7	2.1	0.4 (±0.1)* 1.7 (±0.2)**	6.8	1.6	0.2 (±0.1)* 0.9 (±0.1)**	6.4	1.4	0.7 (±0.0)* 1.3 (±0.3)**

* Change between visit 4 and visit 7
** Total change from baseline to visit 7

Table 4.4.6 Intra-Group Comparison of the Effect on ROMe Scores of Each Intervention Group

Multivariate Tests[b]							
Group	Effect		Value	F	Hypothesis df	Error df	Sig.
Manipulation	time						
		Wilks' Lambda	0.515	9.410[a]	2.000	20.000	0.001
Rehabilitation	time						
		Wilks' Lambda	0.524	7.727[a]	2.000	17.000	0.004
Combination	time						
		Wilks' Lambda	0.403	13.332[a]	2.000	18.000	<0.001

a. Exact statistic
b. Design: Intercept
Within Subjects Design: time

Summary of the outcome scores (excluding WOMAC) between the three intervention groups

Table 4.4.7 summarises the outcomes for each of the outcomes over the visit 1 to visit 4; visit 4 to visit 7 and the visit 1 to visit 7 periods. This summary shows that from the raw scores achieved that the combination group and manipulation group seemed to fair equally well in terms of the numbers of outcomes attained. When read in conjunction with Table 4.4.8 it is confirmed that the BBS, OLST and ROMe did not achieve clinically

significant levels; whereas the ROMf, OTE.1, OTE.2 did achieve a level of clinical significance in all groups. No criteria for BDI was found in the literature.

Table 4.4.7 Summary Of Unit Change In Outcome Scores Between Treatment Groups

		Manipulation	Rehabilitation	Combination
BDI	V1-v4 V4-v7 V1-v7	1.1 1.3 2.4	0.4 0.3 1.0	1.5 0.8 2.3
BBS	V1-v4 V4-v7 V1-v7	3.9 1.1 5.0	1.8 1.2 3.0	2.4 1.1 3.5
OLST	V1-v4 V4-v7 V1-v7	1.4 0.6 2.0	2.8 1.2 4.0	1.4 3.0 4.4
ROMf	V1-v4 V4-v7 V1-v7	10.4 2.9 13.3	7.8 1.1 8.9	7.6 3.8 11.4
ROMe	V1-v4 V4-v7 V1-v7	1.3 0.4 1.7	0.7 0.2 0.9	0.6 0.7 1.3
OTE.1	v4 v4 - v7 v7	4.0 1.4 5.4	3.4 1.0 4.4	3.9 1.7 5.6
OTE.2	V4 V4 – v7 V7	4.4 0.9 5.3	3.6 1.3 4.9	4.5 1.1 5.6

Table 4.4.8
Summary of Minimum Clinically Indicated Change for Each of the Outcomes Used in this Study

Outcome Measure	Percentage/ Unit Change Requires
WOMAC	20%
BDI	No criteria found in literature
BBS	6 point
OTE	14% or 2.2 points
OLST	5 seconds
ROM	5-10°

4.5.2 Inter-group Analysis

4.5.2.1 Subjective Data

4.5.2.1.1 WOMAC

Overall WOMAC Score

There was no statistically significant difference in terms of change of the Overall WOMAC score over time between the three treatment groups (p =0.156). It is, therefore, concluded

that there is not enough evidence to reject the null hypothesis that all three treatments have the same effect in terms of the overall WOMAC outcomes. It is, however, of interest to note the two cross-overs that occur in relation to the Combination group (viz. where the combination group intersects with the other two groups) and the cross-over between the manipulation and the rehabilitation groups. These suggest that an increased sample size may have been required to reach significance.

Table 4.5.1 Inter-Group Comparison of Time Effects for Overall WOMAC Score

Multivariate Tests[c]

Effect		Value	F	Hypothesis df	Error df	Sig.
time * Group						
	Wilks' Lambda	0.891	1.696[a]	4.000	114.000	0.156

a. Exact statistic
b. The statistic is an upper bound on F that yields a lower bound on the significance level.
c. Design: Intercept + Group
Within Subjects Design: time

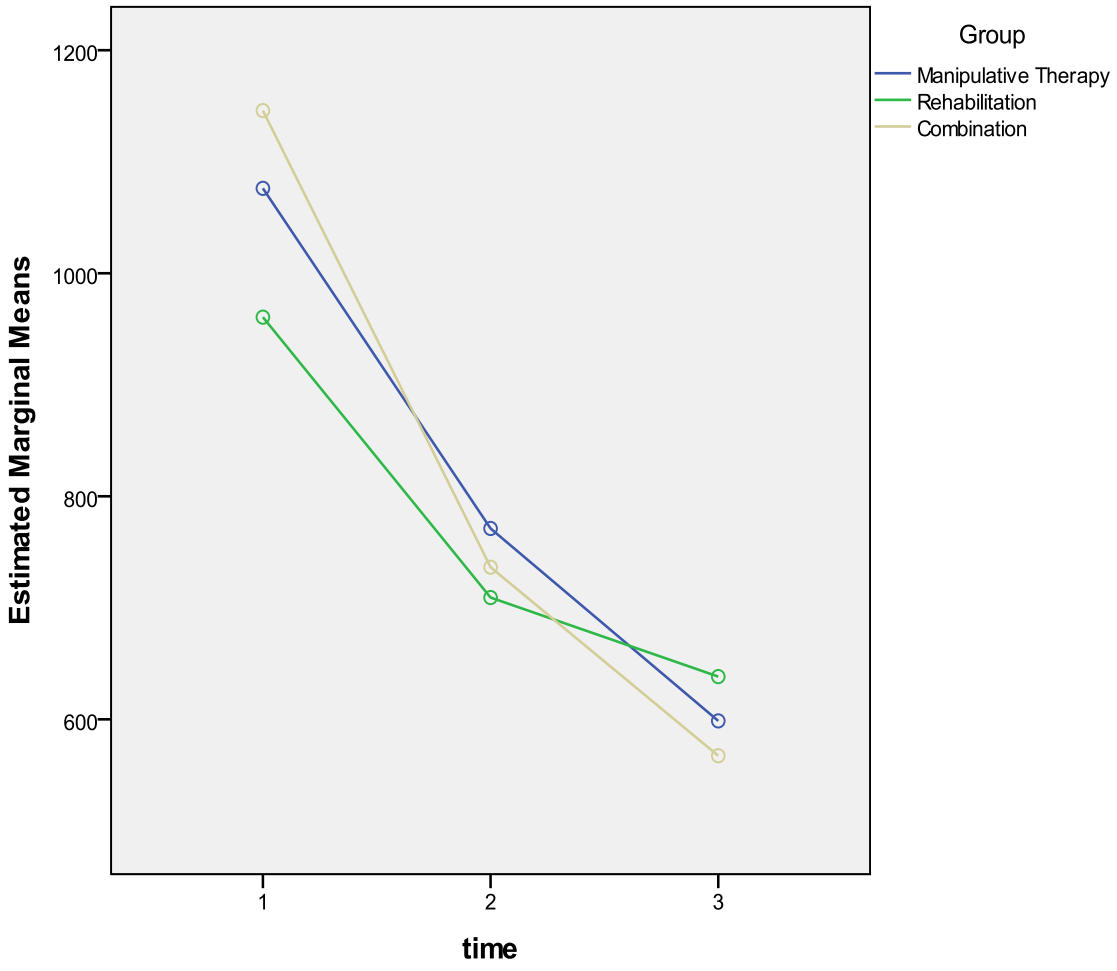

Figure 4.2 Mean Overall WOMAC Score over Time by Treatment Group

WOMAC Pain Score

There was no statistically significant difference in terms of change of WOMAC-P score over time between the three treatment groups (p=0.188). It can, therefore, be conclude that there was not enough evidence to reject the null hypothesis that all three treatment modalities have the same effect. Again attention is drawn to the cross-overs between the manipulation and combination groups which intersect with the rehabilitation group.

Table 4.5.2 Inter-Group Comparison of Time Effects for WOMAC Pain Score

Multivariate Tests[c]						
Effect		Value	F	Hypothesis df	Error df	Sig.
time * Group						
	Wilks' Lambda	0.899	1.566[a]	4.000	114.000	0.188

a. Exact statistic
b. The statistic is an upper bound on F that yields a lower bound on the significance level.
c. Design: Intercept + Group
Within Subjects Design: time

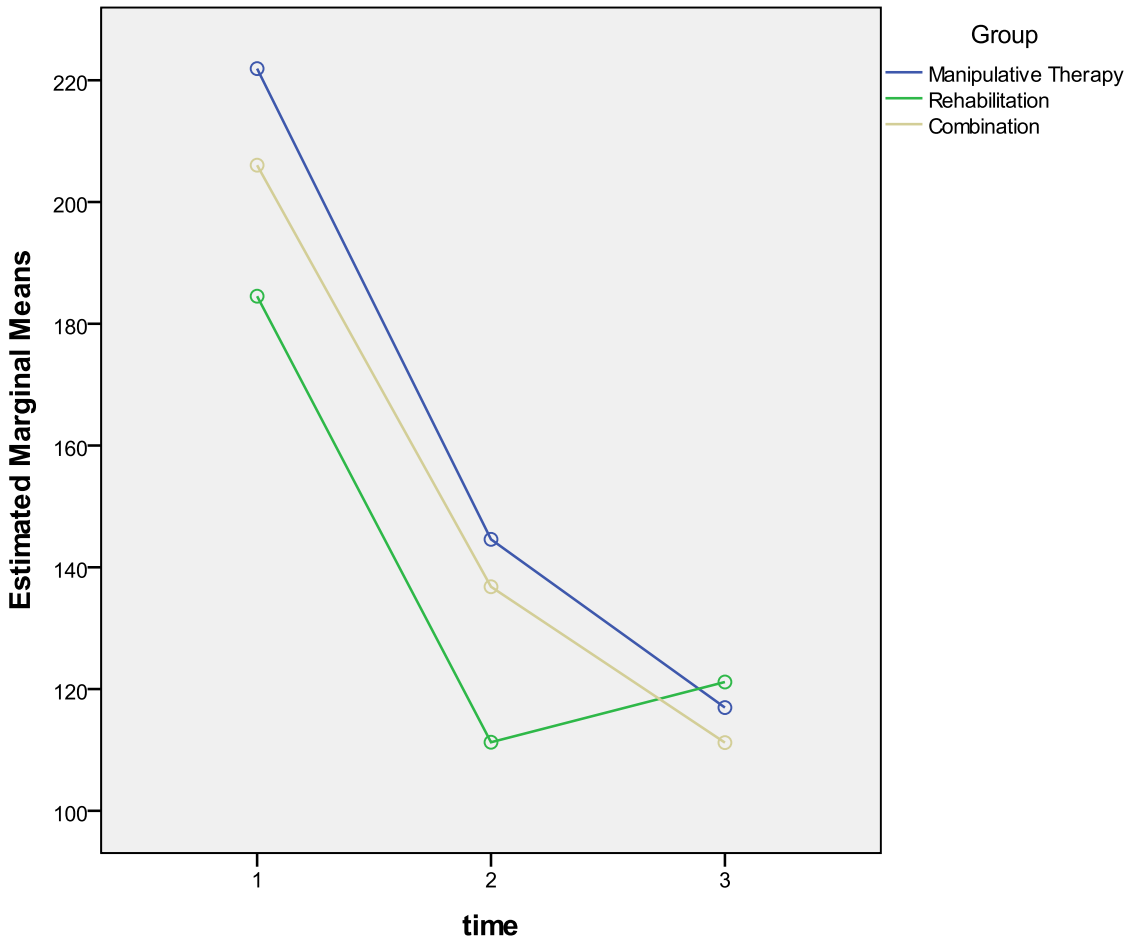

Figure 4.3 Mean WOMAC Pain Score over Time by Treatment Group

WOMAC Stiffness Score

There was no statistically significant difference in terms of change of WOMAC-S score over time between the three treatment groups (p=0.165). It can, therefore, be conclude that there was not enough evidence to reject the null hypothesis that all three treatment modalities have the same effect. The only cross-over that exists in this measure was between the manipulation and the rehabilitation groups.

Table 4.5.3 Inter-Group Comparison of Time Effects for WOMAC Stiffness Score

Multivariate Tests[c]						
Effect		Value	F	Hypothesis df	Error df	Sig.
time * Group						
	Wilks' Lambda	0.893	1.657[a]	4.000	114.000	0.165

a. Exact statistic
b. The statistic is an upper bound on F that yields a lower bound on the significance level.
c. Design: Intercept + Group
Within Subjects Design: time

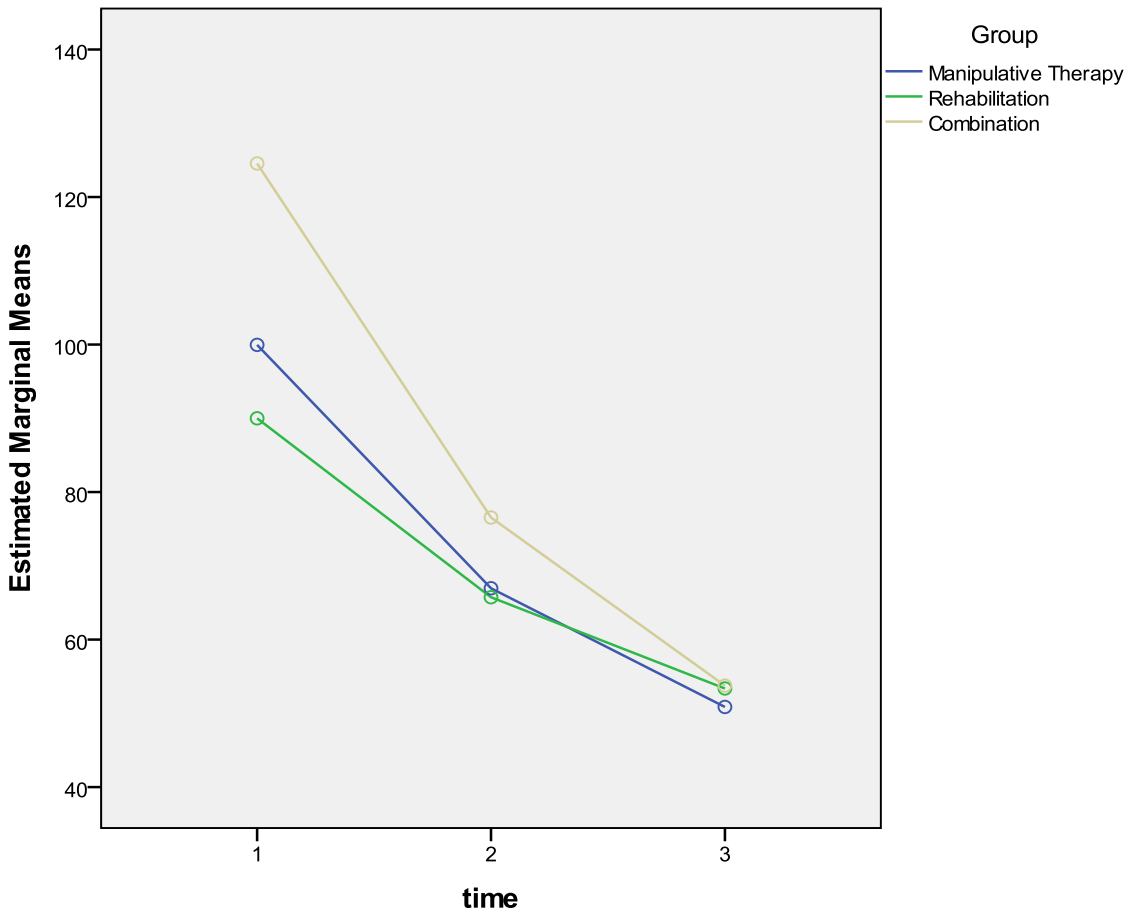

Figure 4.4 Mean WOMAC Stiffness Score over Time by Treatment Group

WOMAC Disability Score

There was no statistically significant difference in terms of change of WOMAC-D score over time between the three treatment groups (p=0.139). It can, therefore, be conclude that there was not enough evidence to reject the null hypothesis that all three treatment modalities have the same effect. A similar pattern of cross-overs is seen here, when compared to the WOMAC overall score.

Table 4.5.4 Inter-Group Comparison of Time Effects for WOMAC Disability Score

Multivariate Tests[c]						
Effect		Value	F	Hypothesis df	Error df	Sig.
time * Group						
	Wilks' Lambda	0.886	1.776[a]	4.000	114.000	0.139

a. Exact statistic
b. The statistic is an upper bound on F that yields a lower bound on the significance level.
c. Design: Intercept + Group
Within Subjects Design: time

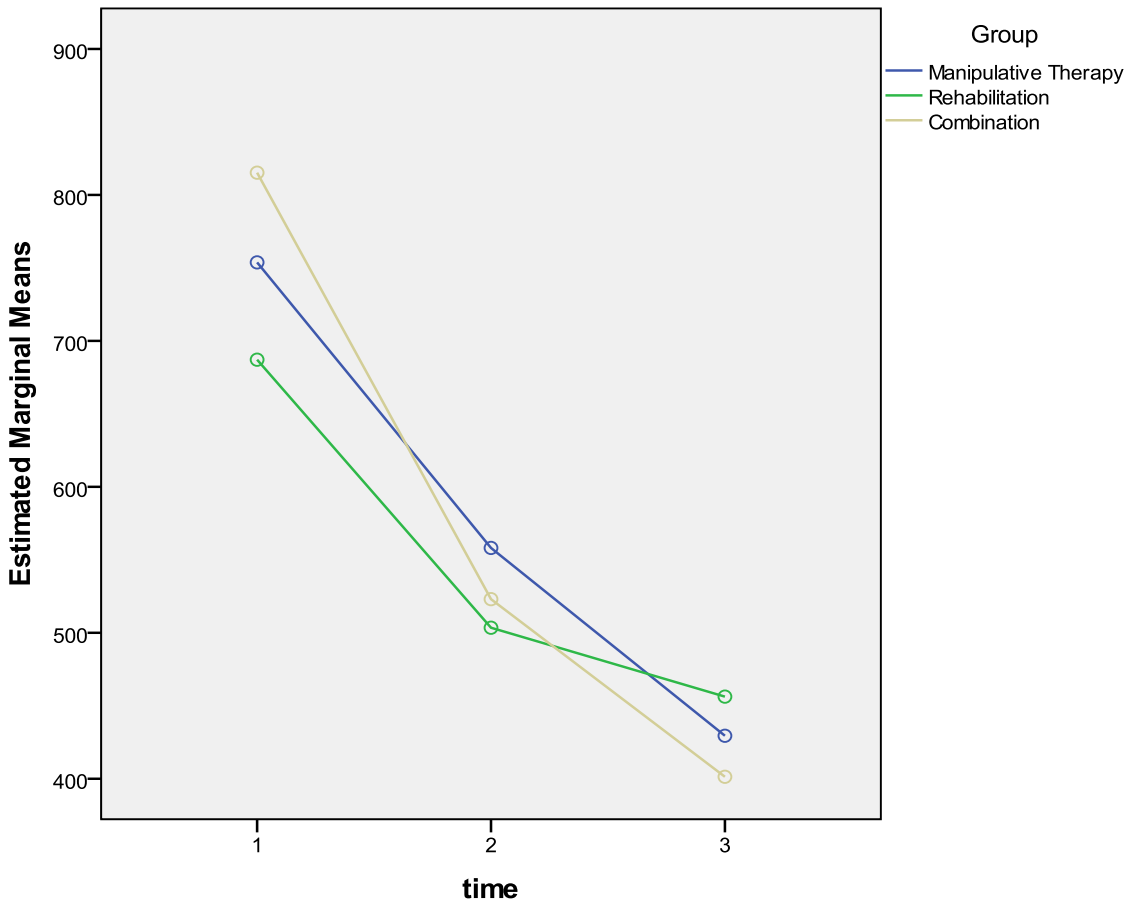

Figure 4.5 Mean WOMAC Disability Score over Time by Treatment Group

4.5.2.1.2 Beck Depression Inventory

There was no statistically significant difference in terms of change of BDI score over time between the three treatment groups (p=0.589). It can, therefore, be conclude that there was not enough evidence to reject the null hypothesis that all three treatment modalities have the same effect. The only cross-over effect is evident at the intersection between the manipulation and rehabilitation groups.

Table 4.5.5 Inter-Group Comparison of Time Effects for BDI

Multivariate Tests[c]						
Effect		Value	F	Hypothesis df	Error df	Sig.
time * Group						
	Wilks' Lambda	0.952	0.706[a]	4.000	114.000	0.589
a. Exact statistic b. The statistic is an upper bound on F that yields a lower bound on the significance level. c. Design: Intercept + Group Within Subjects Design: time						

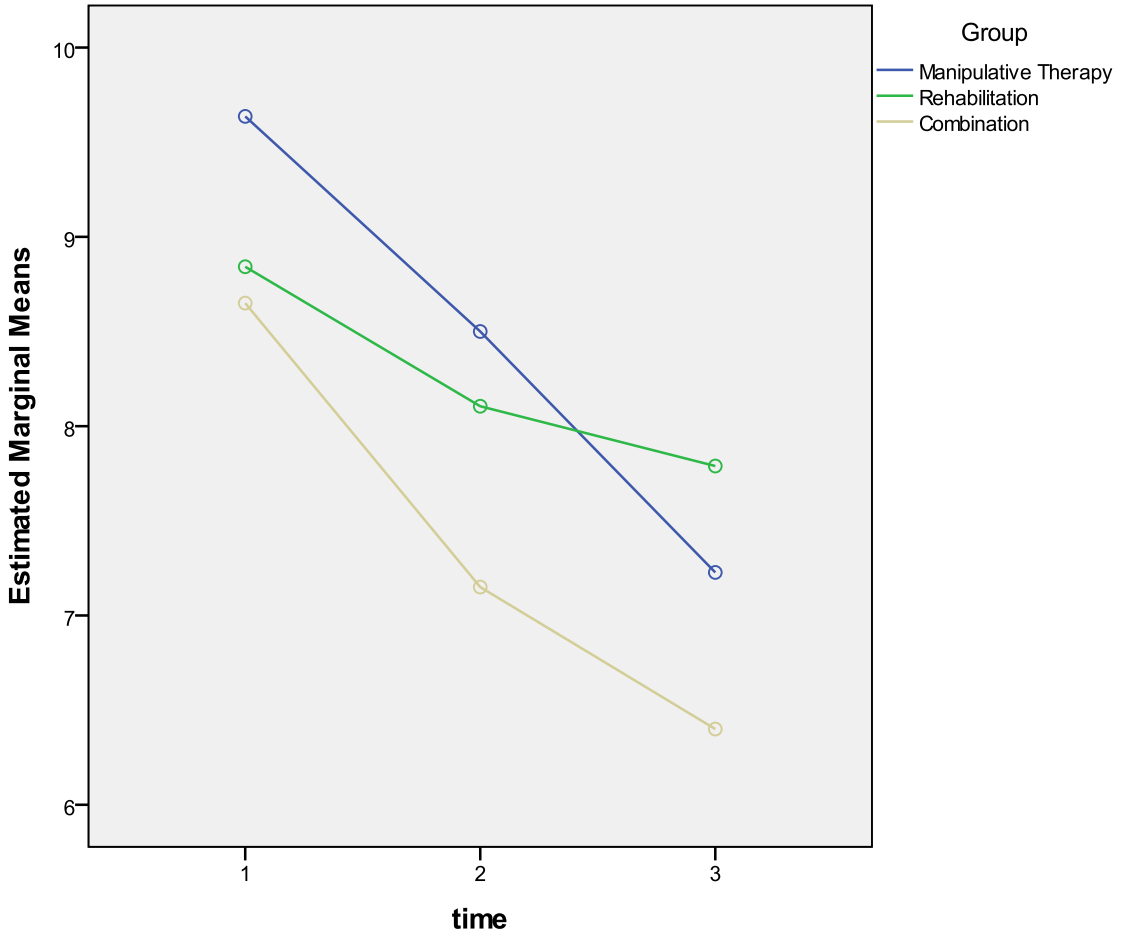

Figure 4.6 Mean BDI Score over Time by Treatment Group

4.5.2.1.3 Berg Balance Scale

There was no statistically significant treatment effect in terms of change of BBS score over time between the three treatment groups (p=0.805). Figure 4.7 shows that the increase over time was uniform in all three groups. The only noted intersection is between the combination group and the rehabilitation group. The manipulation group started at a higher baseline, and therefore, it is likely that an intersection of this and the combination groups would only have been possible if the trial had continued for a longer duration.

Table 4.5.6 Inter-Group Comparison of Time Effects for BBS

Multivariate Tests[c]						
Effect		Value	F	Hypothesis df	Error df	Sig.
time * Group						
	Wilks' Lambda	0.911	0.403[a]	4.000	34.000	0.805

a. Exact statistic
b. The statistic is an upper bound on F that yields a lower bound on the significance level.
c. Design: Intercept + Group
Within Subjects Design: time

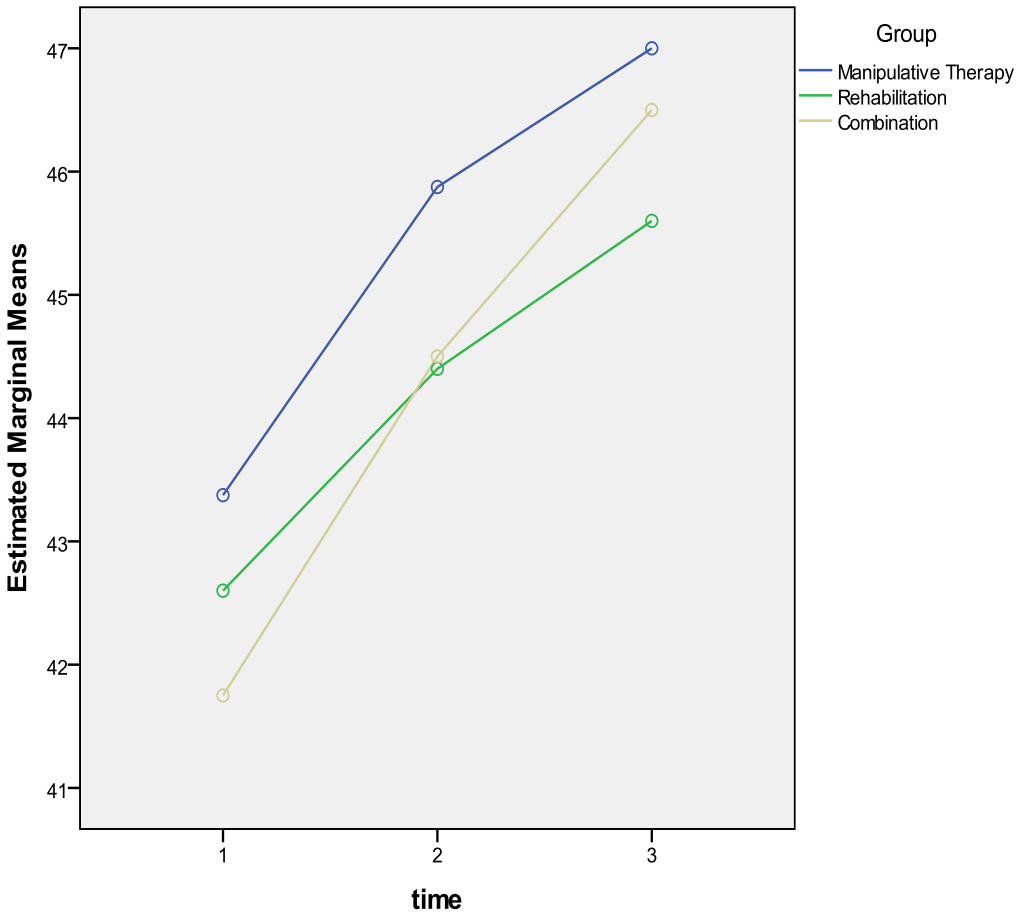

Figure 4.7 Mean BBS Score over Time by Treatment Group

4.5.2.1.4 McMaster Overall Therapy Effectiveness Tool

OTE.1

There was no statistically significant treatment effect for OTE 1 (p=0.690). Figure 4.8 shows that there was a slight trend towards the treatment effect being higher in the combination group but this difference was not statistically significant. A cross- between the combination and the manipulation group is noted for this outcome.

Table 4.5.7 Inter-Group Comparison of Time Effects for OTE.1

Multivariate Tests[b]						
Effect		Value	F	Hypothesis df	Error df	Sig.
time * Group						
	Wilks' Lambda	.987	.373[a]	2.000	58.000	0.690
a. Exact statistic b. Design: Intercept + Group Within Subjects Design: time						

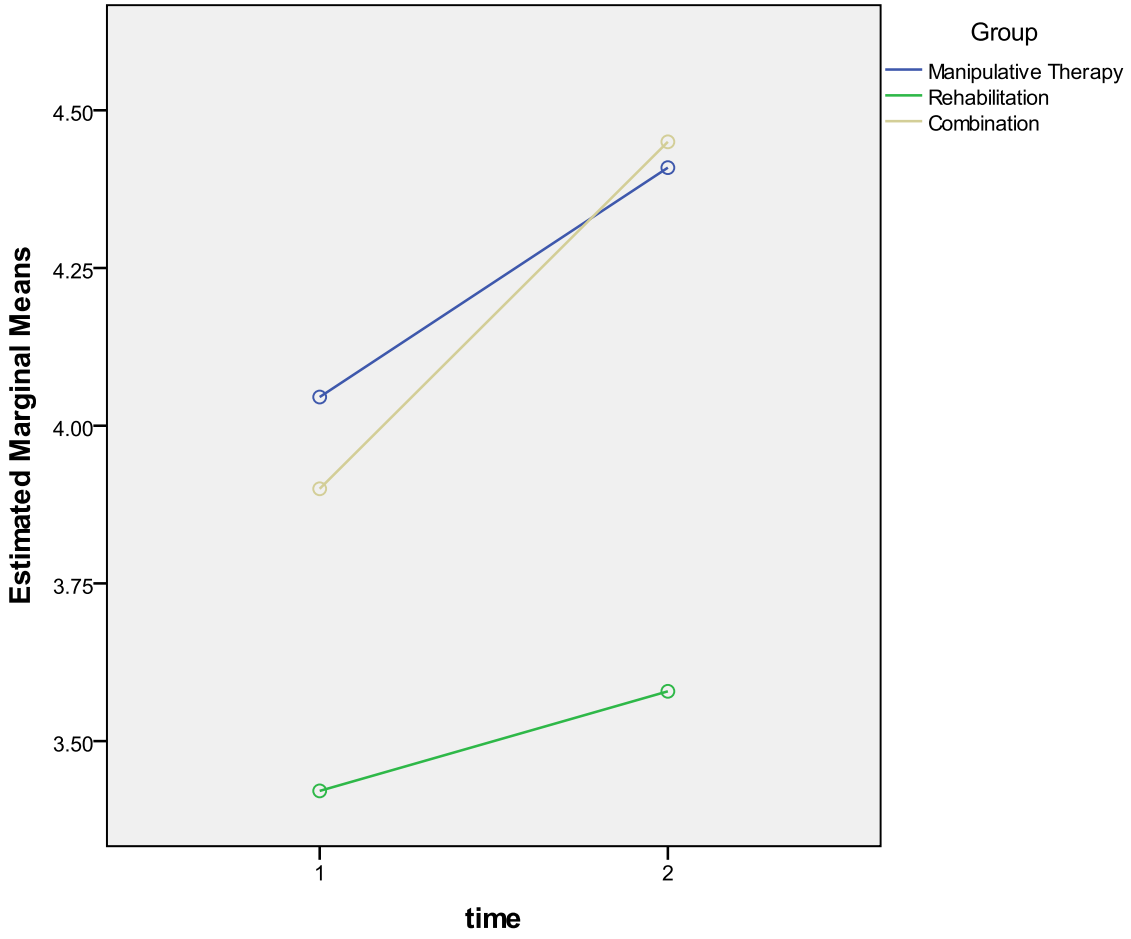

Figure 4.8 Mean OTE.1 Score over Time by Treatment Group

OTE.2

At each time point, the three groups were compared in terms of response to the 2nd part of the OTE questionnaire. At visit 4, there was a significant difference in response between the three groups (p=0.025) but not at visit 7 (p=0.441). Figure 4.9 shows that the combination group experienced the highest scores at both time points.

Table 4.5.8
Kruskal-Wallis Test to Compare Median OTE.2 Scores between the Three Treatment Groups

Ranks				*p*-value
	Group	N	Mean Rank	
OTE1.2	Manipulation	21	32.38	0.025
	Rehabilitation	18	20.92	
	Combination	19	34.45	
	Total	58		
OTE2.2	Manipulation	22	30.48	0.441
	Rehabilitation	17	25.97	
	Combination	20	32.90	
	Total	59		

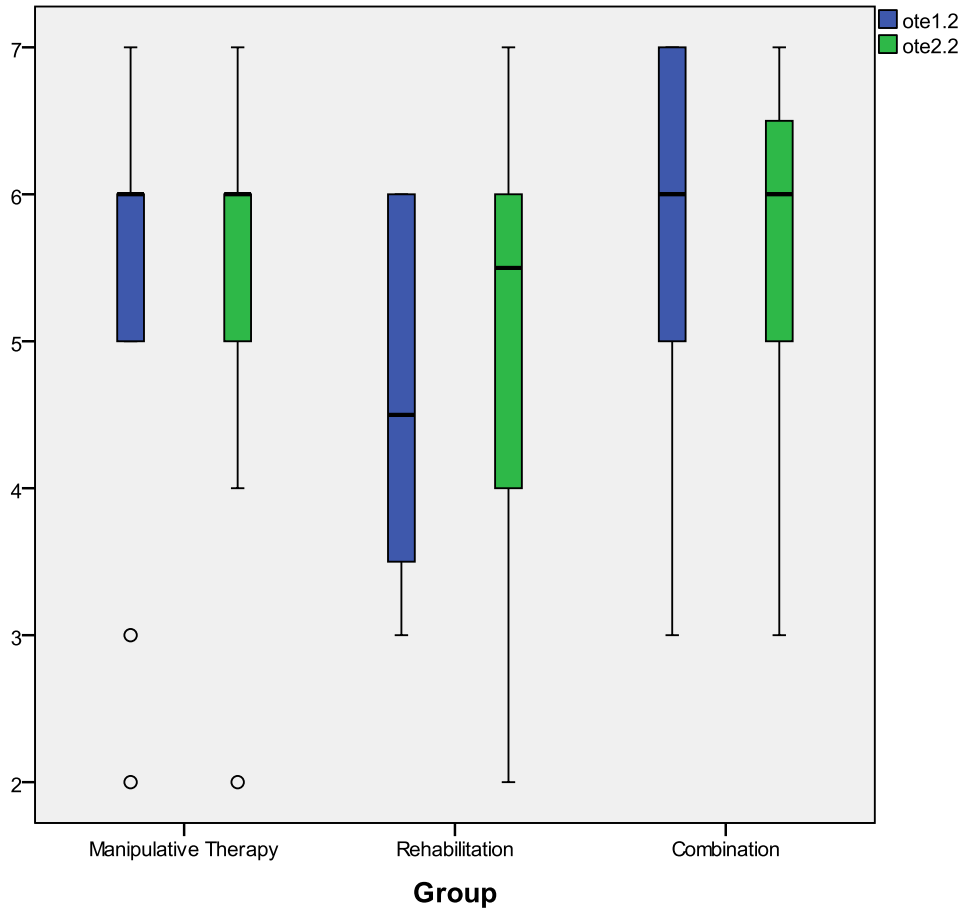

Figure 4.9 Boxplot of OTE.2 Scores by Group end Time Point

4.5.2.2 Objective Data

4.5.2.2.1 One Leg Standing Test

There was a statistically significant intervention effect (p=0.020) meaning that the treatments were not equally effective over time. Examination of Figure 4.10 shows that the combination group showed the highest rate of improvement compared with the other two groups.

Table 4.6.1 Inter-Group Comparison of Time Effects for OLST

Multivariate Tests[c]						
Effect		Value	F	Hypothesis df	Error df	Sig.
time * Group	Wilks' Lambda	.548	3.333[a]	4.000	38.000	0.020

a. Exact statistic
b. The statistic is an upper bound on F that yields a lower bound on the significance level.
c. Design: Intercept + Group
Within Subjects Design: time

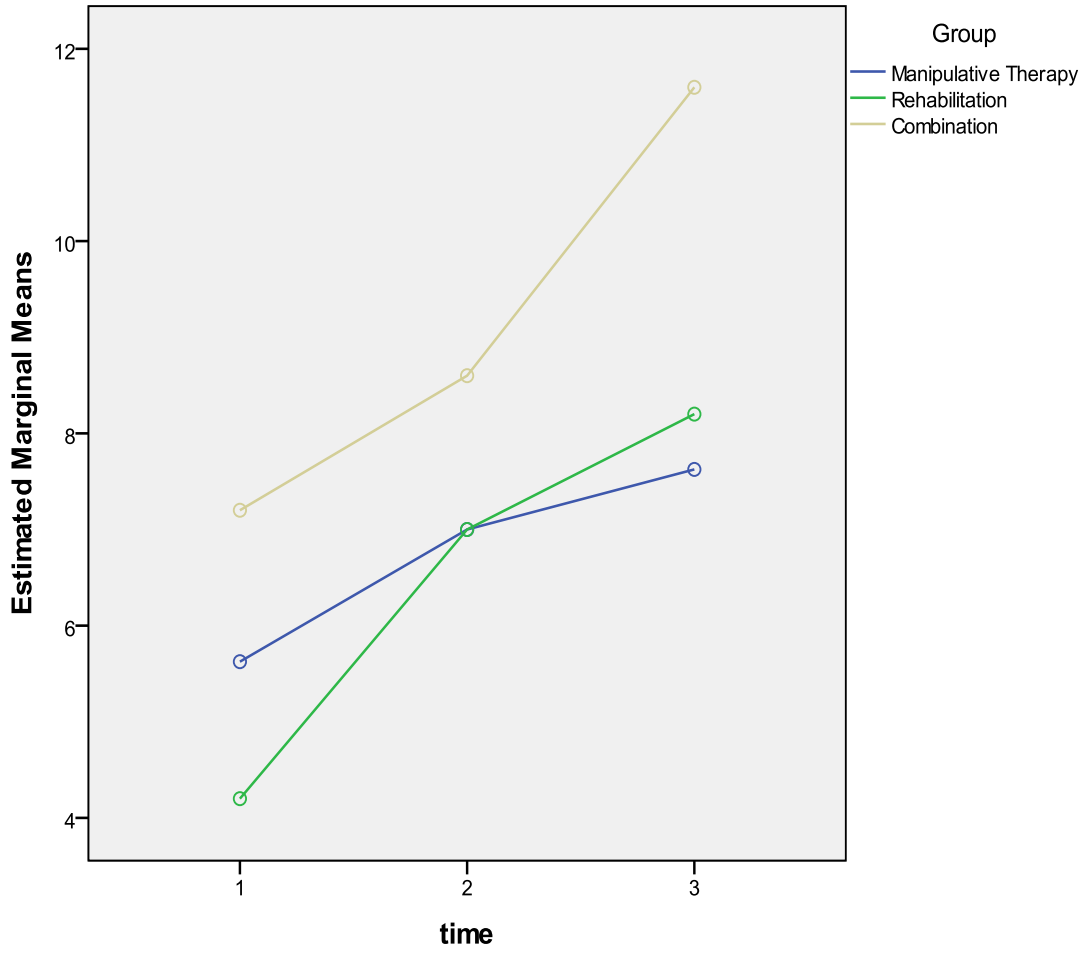

Figure 4.10 Mean OLST Score over Time by Treatment

4.5.2.2.2 Range of Motion

Flexion

There was no significant difference in terms of increase in ROMf value over time between the three treatment groups (p=0.448). It can, therefore, be conclude that there was not enough evidence to reject the null hypothesis that all three treatments have the same effect.

Table 4.6.2 Inter-Group Comparison of Time Effects for ROMf

Multivariate Tests[c]						
Effect		Value	F	Hypothesis df	Error df	Sig.
time * Group	Wilks' Lambda	0.938	0.933[a]	4.000	114.000	0.448

a. Exact statistic
b. The statistic is an upper bound on F that yields a lower bound on the significance level.
c. Design: Intercept + Group
Within Subjects Design: time

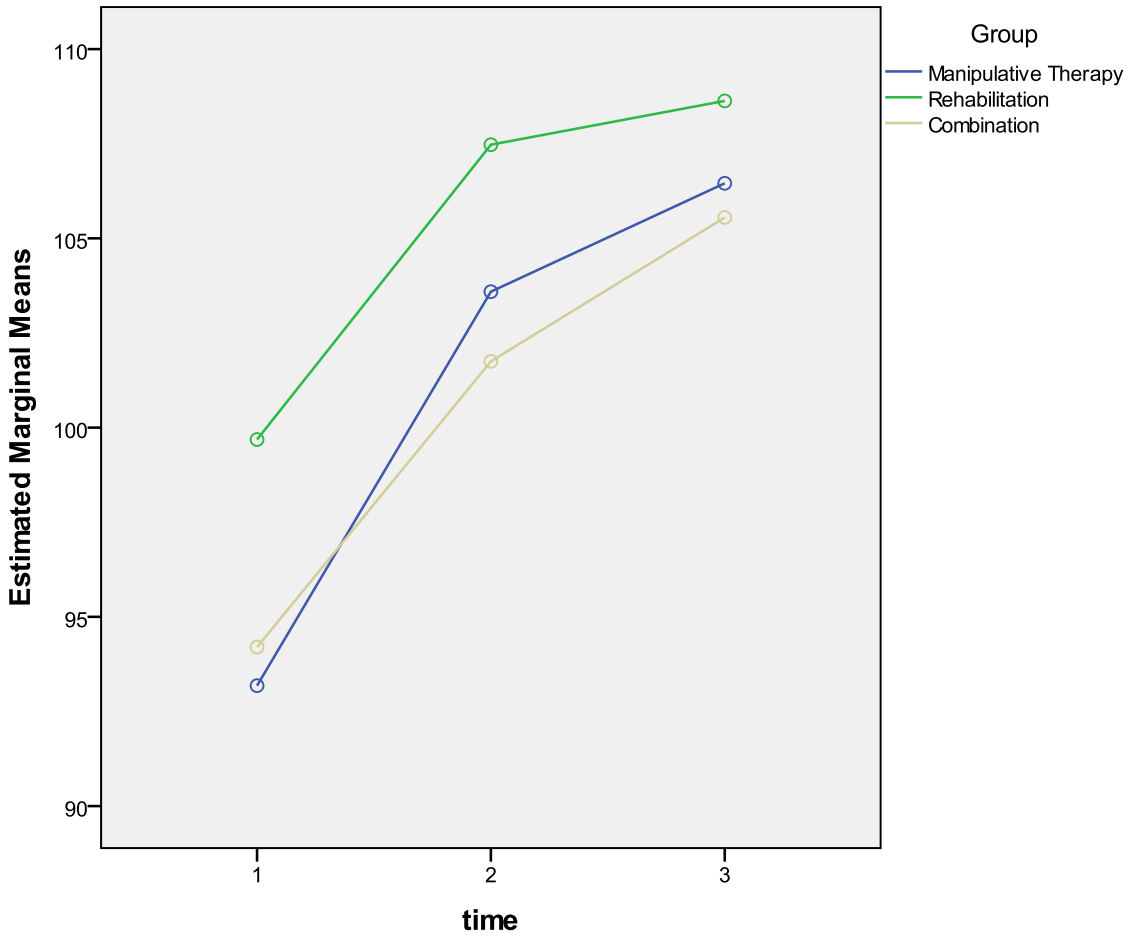

Figure 4.11 Mean ROMf Score over Time by Treatment

Extension

There was no significant difference in terms of increase in ROMe value over time between the three treatment groups (p=0.084). It can, therefore, be conclude that there was not enough evidence to reject the null hypothesis that all three treatments have the same effect.

Table 4.6.3 Inter-Group Comparison of Time Effects for ROMe

Multivariate Tests[c]						
Effect		Value	F	Hypothesis df	Error df	Sig.
time * Group						
	Wilks' Lambda	0.867	2.109[a]	4.000	114.000	0.084

a. Exact statistic
b. The statistic is an upper bound on F that yields a lower bound on the significance level.
c. Design: Intercept + Group
Within Subjects Design: time

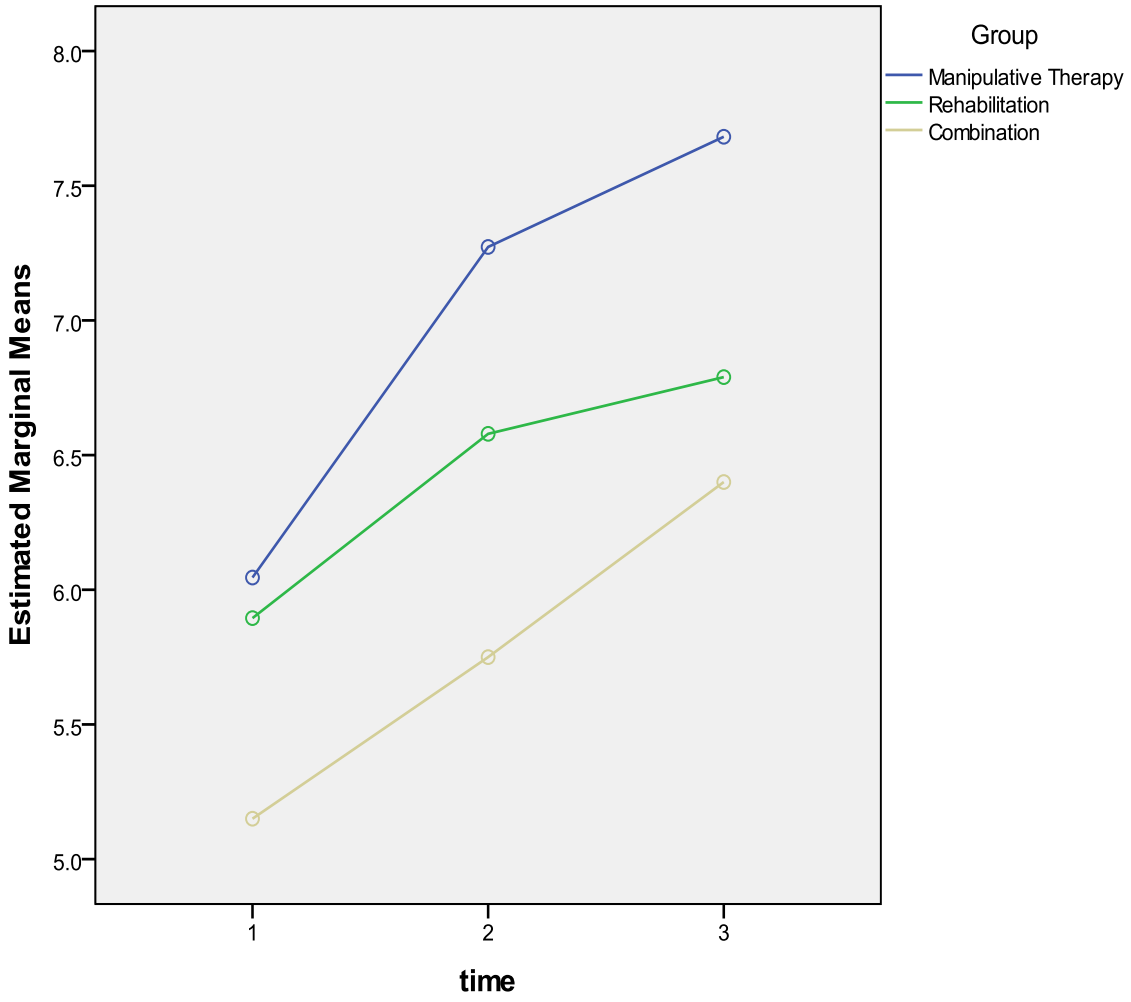

Figure 4.12 Mean ROMe Score over Time by Treatment

4.6 Summary

In terms of the subjective outcomes, all three intervention protocols showed statistically and clinically significant improvements over time for the WOMAC (and its sub-sections). The Beck Depression Inventory and OTE.1 measurements scores were only found to have statistically improved in the combination group; however there was not sufficient improvement to constitute a clinically meaningful change. The OTE.2 measurement score failed to show a statistically or clinically significant improvement in any of the intervention groups.

Objective outcomes (flexion and extension ranges of motion) showed statistically significant improvement over time in all intervention groups; however this improvement was only clinically significant for the ROMf outcome.

The impaired balance subgroup that performed the Berg Balance Scale and One Leg Standing Test at follow-up also showed statistically significant improvement in all three intervention groups across both outcome measures. Inter-group analysis showed the combination group to have the greatest impact on OLST scores (p=0.020). However, this outcome is only reflective of a small portion of the research population, and no conclusions can therefore be drawn about any intervention being superior to the others.

All three intervention protocols showed a statistically significant improvement within the group for most of the subjective and objective outcome measures at the 1-week follow-up. However, when comparing between groups, with the exception of OLST, there was no statistically significant difference and it can, therefore, be conclude that the interventions appear to be equally effective in the short-term management of knee OA.

Table 4.7.1 Summary of the Level of Statistical Significance, Clinical Significance and Overall Change of Outcomes Measures in Each Intervention Group from Baseline to Final Follow-Up.

	Manipulation			Rehabilitation			Combination		
	Change	Statistical Significance	Clinical Significance	Change	Statistical Significance	Clinical Significance	Change	Statistical Significance	Clinical Significance
Overall WOMAC	44.2%	+++	Y	33.6%	++	Y	50.5%	+++	Y
WOMAC Pain	47.3%	+++	Y	34.3%	++	Y	46.0%	+++	Y
WOMAC Stiffness	49.1%	+++	Y	40.7%	++	Y	56.8%	+++	Y
WOMAC Disability	43.0%	+++	Y	33.6%	++	Y	50.8%	+++	Y
BDI	2.4	-		1.0	-		2.3	++	
BBS	5.0	+	N	3.0	+	N	3.5	+	N
OTE.1	5.4	-	N	4.4	-	N	5.6	+	N
OTE.2	5.3	-	N	4.9	-	N	5.6	-	N
OLST	2.0	+	N	4.0	+	N	4.4	+++	N
ROMf	13.3°	+++	Y	8.9°	++	Y	11.4°	+++	Y
ROMe	1.7°	++	N	0.9°	++	N	1.3°	+++	N

+++ if $p < 0.001$
++ if $p = 0.001$- 0.01
+ if $p = 0.01 – 0.05$
- if $p > 0.05$

CHAPTER FIVE

DISCUSSION

5.1 Introduction

This chapter consists of a discussion of the results that were presented in Chapter Four. Firstly the demographics will be presented, followed by the discussion of the subjective and objective data. The results of this study will be compared with the available literature stated in Chapter Two, to determine how the results of this study compare or contrast to the reviewed literature.

5.2 Demographics and Physical Characteristic

Sixty-six patients were divided by concealed allocation into three equal treatment groups, using a computer generated randomisation table [Appendix Q]. Each group included twenty-two subjects who received manipulation, rehabilitation, or a combination of manipulation and rehabilitation.

Of the 66 patients, a total drop-out rate was 19.7%; 5 subjects were excluded prior to visit 4 readings (these patients were replaced), while a further 9 subjects (13.6%) dropped out between the 4th and 7th visit. Missing data (for the 9 subjects that dropped out between visit 4 and 7) were obtained using the intent to treat analysis (a conservative approach was taken in that the intent to treat was maintained at the same level that the patient left the study, without assuming improvement or regression of the patient's condition). Main reasons given for not completing the trial included transport problems or inability to return for follow-up appointments (n= 5), student protest action resulting in temporary closure of the clinic (n=3), or subjects choosing not to continue the research (n=4). Only one subject dropped out as a result of adverse effect of treatment, while another was excluded after sustaining trauma to the knee during activity that was unrelated to the research.

Of the 61 patients whose data was used, 40 (65.6%) were female, 85.2% of whom had an insidious development of symptoms; occurring in the left (n = 20), right (n = 34) or both (n= 7) knees. Most common occupations were pensioners (39.3%), housewives (23.0%), teachers (4.9%), shop workers (4.9%) or domestic workers (3.3%).

Table 4.1.2 shows the even distribution of age, height, and weight of subjects within their respective treatment groups. There was an apparent (but not significant) difference in distribution of duration of symptoms between groups, with the manipulation and combination groups experiencing symptoms for 84.8 (±82) months and 103.6 (±145.3) months, respectively, while the rehabilitation groups had a relatively shorter duration of symptoms, 55.3 (±59.1) months. This difference in means may appear to be large, but due to the very large standard deviations of the groups, the difference between the groups was not statistically significant (p=0.338) (i.e. there was significant overlap in the distributions, making them essentially similar). One may however have expected that the rehabilitation group would have a greater likelihood for improvement based on the relatively short duration of the symptoms. It is therefore recommended that future studies consider making the groups more homogenous and / or stratifying the sample more strictly according to the duration of the symptoms.

Nevertheless, this study population was in keeping with current literature which suggests that age, obesity, female gender, and occupational knee bending are major risk factors for development of OA (Felson, 1990; Cooper *et al*., 2000), and is in keeping with that which is most common, clinically (Boon *et al*., 2006).

5.3 Comparison of Baseline Outcome Measurements

Table 4.2.2 reflects that, with the exception of the WOMAC-Stiffness (WOMAC-S) outcome, all subjects started the research with the same baseline subjective and objective findings.

In terms of the WOMAC-S, there was a statistically significant difference between groups (p=0.040). The Bonferroni post hoc tests showed that rehabilitation and combination groups differed significantly (p=0.043) while none of the other pairs differed. This may have been related to the relative length of time that the patients had had their knee OA (Table 5.1). However, due consideration needs to be given to the fact that the manipulation and combination groups had one (252 months) and two (600 months, 348 months) outlier patients respectively; who modify the median and mean of the groups. Notwithstanding this, it would seem plausible that the younger “pathogenesis” group would potentially be more amenable to an intervention as compared to a group that has had knee OA for many more months or

years (Daniel, Akeson and O'Connor, 1990). However by contrast, it could also be argued that the rehabilitation group had the least ability to improve, in that they had travelled the least down the road of histological and structural changes (Marchiori, 1999; Taylor and Resnick, 2000) when compared to the other two groups.

Table 5.1 Distribution of Sample According to Demographic Characteristics (Quantitative)

	Group								
	Manipulation			Rehabilitation			Combination		
	Median	Mean	SD	Median	Mean	SD	Median	Mean	SD
Age (yrs)	62	63.1	10.8	58.5	62.1	10.8	59	62.1	10.3
Duration (mo)	132	84.8	82.0	126	55.3	59.1	306	103.6	145.3

The effect of the duration of knee OA therefore needs to be taken into account and considered when contextualising the following results which look at the intervention effect.

5.4 Assessment of Intervention Effect

5.4.1 Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC)

The WOMAC is a subjective evaluation in which subjects were required to answer 23 questions regarding symptoms related to knee OA. This chapter will discuss each treatment group's overall WOMAC scores, as well as in part.

Overall WOMAC Score

All three treatment groups showed both clinically and statistically significant changes in overall WOMAC scores from baseline to one-week follow-up. The combination group showed the greatest improvement, with mean score decreasing by 50.5%, as opposed to the 44.4% and 33.6% shown by the manipulation and rehabilitation groups, respectively.

However, when the results were compared between groups, this difference between rate of improvement was not statistically significant (p= 0.156). These results compare favourably with a similar study completed by Deyle *et al*. (2000), where the overall WOMAC scores at 4-weeks had decreased by 51.8% in the group receiving clinic based manipulation and rehabilitation, as compared to the 15.8% improvement in the placebo-group (the age and

pathogenesis of the condition being similar between the groups showing little deviation (Delye *et al*., 2000 – age group (62±10years)). Deyle *et al*. (2005) later compared the combination of in-clinic manipulation and rehabilitation to at home exercises and found that both groups had improved at the 4-week mark by an average of 51.5% and 26%, respectively. Deyle *et al*., (2005) achieved 26% for the home exercise group as compared to this study where the rehabilitation group (based on the same exercise protocol as Deyle *et al*., 2005) achieved 33.6% improvement. This could either be related to patient age or duration of the condition. The outcome differences may have been related to the age of the patients, however Deyle *et al*., (2005) reports a mean age of 62 (± 9) years for their home exercise group, which compares very well with the rehabilitation group in this study. With regards to the duration of the condition (knee OA), it was noted that Delye *et al*., (2005) reported the average duration as approximately 5 years, which makes the duration of symptoms in patients in the home exercise group in their study slightly longer as compared to this study. The influence of this time on the pathogenesis of the condition, patient compliance, patient mobility and other factors on the outcomes of this and the study by Deyle *et al*., (2005) are unclear, but the differences in the outcomes do perhaps warrant further investigation.

WOMAC Pain (WOMAC-P) Score

There was a statistically significant decrease in mean WOMAC-P scores over time for all treatment groups. The manipulation group had the greatest decrease in mean WOMAC-P scores (47.3%), followed by the combination group (46.0%) and rehabilitation group (34.3%). All groups also achieved the 20% MCIC to deem the changes clinically significant.

However, the abovementioned difference in change between groups was not large enough for it to be considered statistically significant (p= 0.188).

It is interesting to note that the rehabilitation group showed the most rapid improvement between baseline and visit 4. However, this improvement was not maintained, and between the 4th and 7th visit the WOMAC-P scores deteriorated by 9.9%.

These results are to be expected as both the combination and the manipulation groups are expected to have similar results based on the effects of manipulation on pain. In this context manipulation increases the stimulation of the Wyke receptors (Leach, 2003; Suter *et al*.,

2000), which is thought to decrease the perception of pain (of joint or muscle (myofascial) origin). The regression or lesser improvement of the combination group, may be related to activity induced knee OA inflammation, which is not induced in the manipulation only group (who did not do exercise in association with their daily routine). Hence over the longer term the manipulation only group would have maintained high stimulation of the mechanoreceptors in the knee modulating pain, whereas this would have been negated by the substance-P and nociceptive stimulation in those patients irritating their knee with exercise in the combination group (Leach, 2003). Additionally the development of myofascial trigger points from exercise may have added to the perceived pain in the combination group (Travell, Simons and Simons, 1999).

This would be applicable to the rehabilitation group only as these patients would have had no stimulation of the Wyke receptors (through manipulation) (Leach, 2003) and should they had any restriction amenable to intervention, these restrictions would have limited the degree to which the exercise would have been able to fully stimulate the Wyke receptors (Sakamoto *et al*., 2001), thus with the exercise inducing inflammation and the lack of stimulation of the Wyke receptors means that these patients would have responded least. Additionally, the development of myofascial trigger points from exercise (Travell, Simons and Simons, 1999) may have added to the perceived pain in the exercise group.

WOMAC Stiffness (WOMAC-S)

The following changes were seen in the WOMAC-S scores: combination group (56.8%), manipulation group (49.1%), and rehabilitation group (40.7%). These changes were considered both statistically and clinically significant within the groups, however inter-group analysis did not show any treatment to be statistically superior (p= 0.165).

In terms of the stiffness, it is likely that the patients reported stiffness as perceived to be related to muscle stiffness (of myofascial trigger points due to muscle disuse (Travell, Simons and Simons, 1999; Chaitow and DeLany, 2005; Ge, 2010)), which means that the patients are likely to be reporting on the extensor mechanism dysfunction as compared to stiffness from the actual knee joint. When one considers this in the context of the findings, then it becomes apparent that the combination group would have been best able to report improvements with the combination of manipulation (Suter *et al*., 1999 and 2000; Hopkins

and Ingersoll 2000; Hillermann *et al*., 2006) and the effect of moderate exercise (Travell, Simons and Simons, 1999; Chaitow and DeLany, 2005; Ge, 2010) superimposed and synergistically negating the effects of the extensor mechanism dysfunction. By contrast the manipulation group only represents the effect of manipulation on the extensor mechanism dysfunction in isolation to exercise. With the exercise group showing benefit for the quadriceps femoris muscle strengthening but no possible mechanism for the reduction in the arthrogenic muscle inhibition and / or the related reactive myofascial trigger point formation, and therefore limited reduction in the extensor mechanism dysfunction (Suter *et al.,* 1999 and 2000; Hillermann *et al.,* 2006).

WOMAC Disability (WOMAC-D)

In each of the treatment groups there was a statistically and clinically significant change in mean WOMAC-D scores over time, with the combination group showing the greatest decrease (50.8%) followed by manipulation (43.0%) and rehabilitation (33.6%).

However this difference in groups was not statistically significant (p= 0.139), and therefore, no one group can be considered superior.

The outcomes of this particular measure would reflect the outcomes as discussed for the WOMAC-S. Principally because the patients are more likely to report muscle stiffness as compared to joint stiffness, particularly in the exercise groups where delayed onset muscle stiffness or increased muscle ability would have been features or sequelae of the interventions.

Therefore within the WOMAC the following is demonstrated:

Table 5.2 Summary of WOMAC Outcomes Results

	Combination	Manipulation	Rehabilitation	
WOMAC Overall	Reflects overall outcome – most improved	Reflects overall outcome- moderate improved	Reflects overall outcome – least improved	Required for optimal outcome in knee OA patients
	Intervention addresses myofascial trigger points and mechanoreceptors	Intervention addresses mechanoreceptors, myofascial trigger points affected secondarily	Intervention addresses myofascial trigger points, limited if any mechanoreceptive effect	Affected by myofascial trigger points and mechanoreceptors

Table 5.2 Summary of WOMAC Outcomes Results continued…

WOMAC Pain	Moderate decrease - sequelae have a cancelling effect	Decrease the most	Decreased the least	
WOMAC Disability	Decrease the most	Moderate decrease	Decreased the least	
WOMAC Stiffness	Decrease the most	Moderate decrease	Decreased the least	

The assertions in Table 5.2 however require verification in future research with particular emphasis on the role of myofascial trigger points and their role in clouding or obscuring the clinical presentation of knee OA. This association may be in part the reason for the inability in clinical medicine to allow for congruence of the clinical symptomatic picture and the radiographic presentation (Dippenaar, 2005). This may also suggest that there are different population subgroups in the knee OA patient pool that would have different clinical prediction rules (Herbert and Fritz, 2012) that could be applied to them, should these individual sub units of patients be identifiable.

Further to the above, it would also be important to run a clinical trial such as this for a longer duration and complete follow-up measures at a time that would be outside of the window in which muscles take to show improvement in strength or ability (Robergs and Roberts, 2000). This would therefore need to include follow-up measurements (if not also interventions) for a period greater than two months (at minimum).

5.4.2 Beck Depression Inventory (BDI)

Only the combination group showed a statistical difference (p= 0.006) in mean BDI scores over time. Linked to the patient perceived changes that are apparent from the discussion in the WOMAC, these results seem to reflect those of the WOMAC-O, WOMAC-D and WOMAC-S. All three of these factors may be related to the patient perception of ability, with improved ability being related to a decreased Beck Depression Inventory score (Wolfe, Lichtenstein, and Singh, 1999). It cannot be excluded that the duration of symptoms may also have influenced this outcome with the patients that had the most chronic knee OA (longest duration of symptoms) being in the combination group and the patients with the shortest duration of symptoms being in the rehabilitation group.

It is, therefore, suggested the future research look at the association of knee OA duration with clinical outcomes measure (clinical prediction rules (Herbert and Fritz, 2012)) and then also how these outcomes link or do not link with the Beck Depression Inventory for specific durations of knee OA.

5.4.3 Berg Balance Scale (BBS)

It is important to note that not all patients completed the Berg Balance Scale, with only ten in the combination group, eight in the manipulation group and five in the rehabilitation group. This was because these patients failed the OLST at the initial visit. With this in mind, this subgroup in the patient groups had statistically significant improvements, with the manipulation groups showing the greatest improvement (5 point change), followed by the combination (3.5 points) and rehabilitation groups (3.0 points). MCIC was set at 6 points (Stevenson, 2001), and therefore, no group achieved this outcome clinically. Additionally, there was no statistically significant change between groups (p=0.805). It is, however, worth noting that at visit 7 the mean total for the BBS was >45 point, which indicated that those patients who previously failed the BBS no longer represented an increased risk of a fall

In a study of the Berg Balance Scale's effectiveness in predicting a risk of a fall, a nonlinear association between drop in BBS score and risk or fall was noted. A score of 56 is associated with a 10% predicted probability of fall, with those in the 56 to 54 point range, having a 3% to 4% increased risk of fall with every 1-point drop in score. In the 54 to 46 range, a 1-point drop led to a 6% to 8% increased risk, with those scoring below 36 having a predicted risk of fall of almost 100% (Shumway-Cook *et al*., 1997).

Current literature has mixed results regarding the effectiveness of MMT in improving balance. One study found that an eight week course of Chiropractic treatment and supervised exercise resulted in all participants improving their BBS scores above the increased risk threshold (Hawk *et al*., 2007). However, the sample size for this study was relatively small, and when expanded to a larger treatment group, the previously seen benefits failed to be evident (Hawk, Cambron and Pfefer, 2009).

Notwithstanding the above literature, it is possible that these subgroups may also have represented a group effect, as balance is both perceived (myofascial) and actual

(proprioceptive). Therefore, it is possible that the inventions as discussed in Table 5.2 would have a similar effect on the Berg Balance Scale as compared to WOMAC for each group.

5.4.4 The McMaster Overall Therapy Effectiveness Tool (OTE)
Overall Treatment Evaluation (OTE.1) and Overall Treatment Effect (OTE.2)

A statistically significant change was only noted in the combination group (p=0.045) (OTE.1) when comparing effect over time between time points. This indicated that there was change and that it was significantly better than the other groups. This trend is in line with the WOMAC outcomes and the Beck Depression Inventory (Section 5.4.1 and 5.4.2).

Additionally, the patients with the longest duration of symptoms had the greatest improvement which is in line with the suggestion by Tubach *et al*. (2005), who indicated that patients that have the most severe symptoms have to experience a greater change to consider themselves improved.

5.4.5 One Leg Standing test (OLST)

Only the combination group managed to achieve a statistically significant (p<0.001) change in OLST time, which may be related to:

- The fact that they had an increased number of patients that failed the OLST at the outset.
- They had the patients with the longest duration of the knee OA as a complaint and therefore the greatest chance of improvement.
- The possibility that receiving both interventions allowed for a greater effect on proprioception (Leach, 2003), muscle related instability (e.g. “knee giving way) (Travell, Simons and Simons, 1999; Chaitow and DeLany, 2005)

It is, however, noted that, despite the improvement (4.4 ±0.4 seconds), literature suggests that a clinically significant change is greater than 5 seconds increase in time (Hawk and Cambron, 2009). Therefore, it is suggested an increased sample size would be appropriate to detect clinically significant changes and that this may also be related to the possibility of how the patient perceived pain (of joint or muscle (myofascial origin)) and how they reacted to it.

Thus again it is noted that patient sub-grouping (Herbert and Fritz, 2012) in knee OA may be beneficial to improve clinical outcomes.

This latter suggestion is supported by Hawk *et al*. (2007), who found a poor correlation between increased risk of a fall as per a BBS score and OLST (i.e. individuals scored >45 points on BBS yet <5 seconds on OLST). Additionally, OLST failed to reflect retest improvements when scores from the BBS did.

5.4.6 Range of Motion Flexion (ROMf)

Each of the treatment groups showed an improvement in flexion that was considered to be both statistically and clinically significant.

The manipulation appeared to have the greatest increase in range of motion by the seventh visit (13.3°), followed by the combination (11.4°) and rehabilitation (8.9°) groups. However, this difference between groups was not sufficient to reject the null hypothesis (p=0.448).

Additionally, it is noted that these results reflect the outcomes of pain, where the combination intervention group effect was seen to have a cancelling effect, thereby limiting pain and thus by the same mechanism limiting the effect of overall ROM flexion by increasing the stimulus for inflammation by means of the rehabilitation, which limits the motion made available by the manipulation. This is not possible in the manipulation only group which does not have additional rehabilitation. However, the rehabilitation only group is not able to overcome any restrictions in motion within the manipulation intervention, and therefore, cannot improve to the level of the other two groups.

These changes noted were far greater than those seen in previous studies which focus on isolated treatment to the knee joint alone (Tucker, Brantingham and Myberg, 2003; Fish *et al*. (2008). Tucker, Brantingham and Myberg (2003) found in their study that manipulation of the knee joint only resulted in a 1.8° improvement in flexion after eight treatments, while the results from the Fish *et al*. 2008 study showed the mobilisation plus Capsaicin cream group had the highest ROMf increases (5°). The improvements may be related to the fact that the

patients in this study received FKC manipulation, as opposed to the single joint manipulations shown by Tucker, Brantingham and Myberg (2003) and Fish *et al*. (2008).

Extension (ROMe)

All treatment groups saw statistically significant improvements in extension over time, with the manipulation group showing the greatest increase from baseline (1.7°). This was followed closely by the combination (1.3°) and rehabilitation (0.9°) groups. Because of the similarity in rate of change, there was no statistically significant difference between groups (p=0.084). Furthermore, none of the groups had large enough improvements to be deemed clinically significant (as the improvements were too small).

5.4.7 Summary of Findings

Table 5.3 summarises the findings for this study showing possible associations between the different factors. This study could be used as a basis for developing clinical prediction rules.

Table 5.3 Summary of Subjective and Objective Outcomes

	Combination	Manipulation	Rehabilitation
Commonalities possibly associated with a larger myofascial component, increased duration of knee OA or patient perception of change due to length of duration of knee OA			
WOMAC Overall	Reflects most improved overall outcome	Reflects moderate improved overall outcome	Reflects least improved overall outcome
	Intervention addresses myofascial trigger points and mechanoreceptors	Intervention addresses mechanoreceptors, myofascial trigger points affected secondarily	Intervention addresses myofascial trigger points, limited if any mechanoreceptive effect
WOMAC Disability	Decreased the most	Moderate decrease	Decreased the least
WOMAC Stiffness	Decreased the most	Moderate decrease	Decreased the least
BDI	Decreased the most	Moderate decrease	Decreased the least
OTE	Decreased the most	Moderate increase	Decreased the least
Commonalities possibly associated with a larger joint involvement component, decreased duration of knee OA or patient perception of change due to length of duration of knee OA			
WOMAC Pain	Moderate decrease - sequelae have a cancelling effect	Decreased the most	Decreased the least
ROMf	Moderate increase	Increased the most	Increased the least
ROMe	Moderate increase	Increased the most	Increased the least

5.5 Study limitations

Statistically significant changes between groups may have been noted had there been a larger sample size.

A longer follow-up period may have been required to determine which intervention had the greater lasting effect after the treatment period had concluded. Similarly, a long-term study would have better controlled the influence that the natural history of OA has on the clinical presentation, and in turn outcomes measures.

Due to the chronic nature of OA, it is possible that the three week treatment period was not long enough to produce a clinical effect on a long-standing condition. Therefore, it is recommended for further research that those patients who fail to reach a clinically significant change in outcomes measures be places in a “non-responders” groups and receive a further three weeks of treatment. This would assist the researcher in determining whether duration of treatment would influence the results produces.

This was a pragmatic study, in that it contained a lot a variable in selecting which aspect of the kinematic chain to address, based on the patient’s clinical presentation. Motion palpation and manipulation techniques also have a low level of inter-examiner reliability. Therefore, in order for future research to have results that are easily reproducible, it is recommended that the manual therapy protocol be more clearly defined and less patient-specific.

A relatively conservative approach was taken when performing the “Intent To Treat” analysis, whereby data from patients who dropped out after the 4th visit was maintained at the same level that the patient left the study, without assuming improvement or regression of the patient’s condition. Stronger statistical data may have been possible had a multiple imputations approach been used (making the assumption of sustained improvement).

Every attempt was made to control the effect that the patient-practitioner interaction had on the follow-up readings (e.g. use of a blind assessor). However, the possibility of influence by the Hawthorne effect cannot be entirely excluded.

CHAPTER SIX

CONCLUSIONS AND RECOMMENDATIONS

6.1 CONCLUSIONS

This study showed that there was a significant improvement in all three treatment groups with regard to reduction in the symptoms of knee OA. The combination group was most effective with regard to the WOMAC (Overall, as well as WOMAC-S, and WOMAC-D subsets), OTE and BDI outcomes.

However, the manipulation only Group seemed to have greater improvements in range of motion, and decreased pain (as per WOMAC-P). The suspected reason being that the addition of rehabilitation to a manipulation protocol may have aggravated the knee, causing stimulation of nociceptors and release of substance-P (Leach, 2003). This activity induced inflammation would have negated the analgesic effect of manipulation (Suter *et al*., 2000; Hillermann *et al*., 2006). This assertion however would require further investigation in order to verify this physiological explanation.

However, this research paper was unable to demonstrate that a combination of manipulation and rehabilitation had a statistically significant effect over manipulation or rehabilitation alone.

6.2 RECOMMENDATIONS

1) Due to the chronicity of knee OA and its changing clinical picture as the pathogenesis of the disease continues, it is recommended that a long term follow-up be conducted to evaluate the intermediate and long-term effect of the treatment protocol. This would be of particular importance when utilising rehabilitation as a treatment intervention as improvement in muscle strength and ability is reported to only have clinical significant after six weeks (Robergs and Roberts, 2000).

2) Further studies may benefit from a larger sample size in order to improve statistical data.

3) A study that allowed for off-campus treatment to occur would be likely to have a lower drop-out rate as difficulty attending all follow-up appointments due to a lack of transport or the unexpected closure of the clinic during student protest action was the reason given by 43% of the patients who did not complete the trial to the 1-week follow-up mark.

4) Future studies should note the activity level of patients at baseline, as well as develop an accurate measure to determine the degree of compliance to the rehabilitation protocol in order to draw possible comparisons between patients' improvements. This would also facilitate in address the assertion that the ability to perform increased activity in the combination group may be linked to a decrease in clinical outcomes as compared to manipulation alone.

5) It was suspected that the results of this study may have been influenced by the duration the patients had experienced their knee OA symptoms. Thus, it is recommended that further studies attempt to stratify treatment groups by symptom or presentation in order to ensure each group is homogeneous.

6) Furthermore, this research used multiple subjective outcome measures to determine improvement in the patients. It would be recommended that a set of clinical predictive rules (Herbert and Fritz, 2012) be developed to determine how the changes in the various outcome measures may be influenced by the duration of the condition, as well as their mental state (as assessment by the BDI) at the time of evaluation.

7) This study revealed a possible confounding effect that myofascial trigger points (MTPs) may have on the clinical presentation of knee OA. While there is literature to support the presence of muscular changes in knee OA, there is an apparent paucity of knowledge regarding the effect of these changes on pain presentation, activity limitation and other symptoms of knee OA (such as stiffness and decreases range of motion) which are typically associated with articular changes. It is therefore recommended that further studies investigate the role that MTPs have to play in altering the early pathogenesis and presentation of the pathology, as well as the validity of currently used outcome assessments in light of MTP presence.

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APPENDIX A
ADVERTISEMENT

Do you suffer from

ARTHRITIS OF THE KNEE

and aged between

38 and 80?

Research is currently being carried out at the

Durban University of Technology
Chiropractic Day Clinic

FREE TREATMENT

is available for those who qualify for this study on osteoarthritis of the knee.

For further information contact:

Lauren on: 031 373 2205/2512

or 073 153 1435

APPENDIX B

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 C

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 D

REGIONAL EXAMINATION - LUMBAR SPINE AND PELVIS

Patient:_____ File#:_____Date:_________

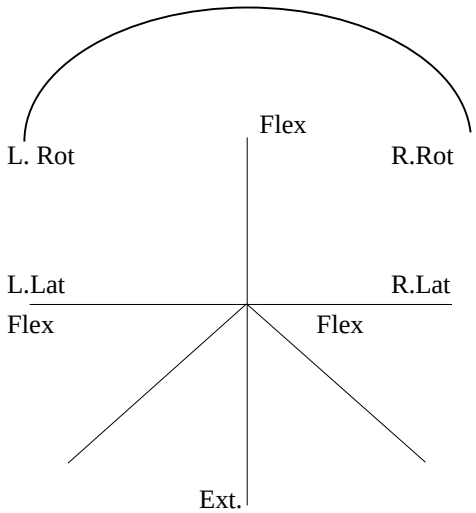
Intern\Resident:_____ Clinician:_____

STANDING:

Posture– scoliosis, antalgia, kyphosis
Body Type
Skin
Scars
Discolouration
Minor's Sign
Muscle tone
Spinous Percussion
Scober's Test (6cm)
Bony and Soft Tissue Contours

GAIT:

Normal walking
Toe walking
Heel Walking
Half squat



ROM:

Forward Flexion = 40-60° (15 cm from floor)
Extension = 20-35°
L/R Rotation = 3-18°
L/R Lateral Flexion = 15-20°

Which movt. reproduces the pain or is the worst?

- Location of pain
- Supported Adams: Relief? (SI)
- Aggravates? (disc, muscle strain)

SUPINE:

Observe abdomen (hair, skin, nails)
Palpate abdomen\groin
Pulses - abdominal
- lower extremity
Abdominal reflexes

		Degree	LBP?	Location	Leg pain	Buttock	Thigh	Calf	Heel	Foot	Braggard
SLR	**L**										
	R										

	L	**R**
Bowstring		
Sciatic notch		
Circumference (thigh and calf)		
Leg length: actual -		
apparent -		
Patrick FABERE: pos\neg – location of pain?		
Gaenslen's Test		
Gluteus max stretch		
Piriformis test (hypertonicity?)		
Thomas test: hip \ psoas? \ rectus femoris?		
Psoas Test		

SITTING:

Spinous Percussion Valsalva

Lhermitte

TRIPOD Sl, +, ++		Degree	**LBP?**	**Location**	**Leg pain**	**Buttock**	**Thigh**	**Calf**	**Heel**	**Foot**	**Braggard**
	L										
	R										

Slump 7 test	**L**										
	R										

LATERAL RECUMBENT: L R

Ober's		
Femoral n. stretch		
SI Compression		

PRONE: L R

Gluteal skyline		
Skin rolling		
Iliac crest compression		
Facet joint challenge		
SI tenderness		
SI compression		
Erichson's		
Pheasant's		

MF tp's	**Latent**	**Active**	**Radiation**
QL			
Paraspinal			
Glut Max			
Glut Med			
Glut Min			
Piriformis			
Hamstring			
TFL			
Iliopsoas			
Rectus Abdominis			
Ext/Int Oblique muscles			

NON ORGANIC SIGNS:

Pin point pain
Trunk rotation
Flip Test
Ankle dorsiflexion test
Axial compression
Burn's Bench test
Hoover's test
Repeat Pin point test

NEUROLOGICAL EXAMINATION

Fasciculations

Plantar reflex

level	Tender?	Dermatomes		DTR		
		L	R		L	R
T12				Patellar		
L1				Achilles		
L2						
L3				Proproception		
L4						
L5						
S1						
S2						
S3						

MYOTOMES

Action	Muscles	Levels	L	R	
Lateral Flexion spine	Muscle QL				
Hip flexion	Psoas, Rectus femoris				5+ Full strength
Hip extension	Hamstring, glutes				4+ Weakness
Hip internal rotat	Glutmed, min;TFL, adductors				3+ Weak against grav
Hip external rotat	Gluteus max, Piriformis				2+ Weak w\o gravity
Hip abduction	TFL, Glut med and minimus				1+ Fascic w\o gross movt
Hip adduction	Adductors				0 No movement
Knee flexion	Hamstring,				
Knee extension	Quad				W - wasting
Ankle plantarflex	Gastroc, soleus				
Ankle dorsiflexion	Tibialis anterior				
Inversion	Tibialis anterior				
Eversion	Peroneus longus				
Great toe extens	EHL				

BASIC THORACIC EXAM

History
Passive ROM
Orthopedic

BASIC HIP EXAM

History
ROM: Active
Passive : Medial rotation : A) Supine (neutral) If reduced - hard \ soft end feel
B) Supine (hip flexed): - Trochanteric bursa

MOTION PALPATION AND JOINT PLAY

	L	R
Upper Thoracics		
Lumbar Spine		
Sacroiliac Joint		

APPENDIX E

HIP REGIONAL EXAMINATION

Patient: _____ File no: _____ Date: _____

Student: _____ Signature: _____

Clinician: _____ Signature: _____

Hip with complaint: Right ☐ Left: ☐

OBSERVATION

- Gait: _____
- Posture: _____
- Weight-bearing symmetry: _____
- Balance and proprioception (Stork-standing test): _____
- Bony / soft tissue contours: Buttock contour _____
  Hip flexion contracture _____
  Lumbar lordosis _____
  Scoliosis _____
- Skin: _____
- Swelling: _____

PALPATION

· **Anterior aspect**			Right	Left
1.	Iliac crests			
2.	Greater trochanter			
3.	Pubic symphysis and tubercle			
4.	Femoral head			
5.	Femoral Δ	Femoral artery		
		Lymph nodes		
6.	ASIS's			
7.	Inguinal ligament			
8.	Inguinal hernia			
9.	Muscles -	Quadriceps		
		Adductors		
		Abductors		
		Psoas		

· **Posterior aspect**			Right	Left
1.	Iliac crests posteriorly			
2.	Ischial tuberosity			
3.	Muscles	Piriformis		
		Gluteals		
		Hamstrings		
4.	PSIS's			
5.	Sciatic notch			
6.	SI joints			
7.	Lumbar Spine			
8.	Sacrum + coccyx			

ACTIVE MOVEMENTS (*note rom and pain*)		Right	Left
1.	Flexion (110-120°)		
2.	Extension (10-15°)		
3.	Adduction (30°)		
4.	Abduction (30-50°)		
5.	Medial rotation (30-40°)		
6.	Lateral rotation (40-60°)		

PASSIVE MOVEMENTS (*note end-feel, rom and pain*)			Right	Left
1.	Flexion (tissue stretch or approximation)			
2.	Extension (tissue stretch)			
3.	Adduction (tissue stretch or approximation)			
4.	Abduction (tissue stretch)			
5.	Medial rotation (tissue stretch)			
6.	Lateral rotation (tissue stretch)			
RESISTED ISOMETRIC MOVEMENTS (*note strength and pain*)			Right	Left
1.	Hip Flexion			
2.	Hip Extension			
3.	Adduction			
4.	Abduction			
5.	Medial rotation			
6.	Lateral rotation			
7.	Knee flexion			
8.	Knee extension			
REFLEXES			Right	Left
1.	Patella			
2.	Achilles			
DERMATOMES (*indicate deficits by level & location*)				
1.	Level			
2.	Location			
JOINT PLAY MOVEMENTS			Right	Left
1.	Caudal glide (long axis traction superior – inferior)			
2.	Compression@ 90^{o} (inferior – superior)			
3.	Medial ➢ lateral @ 180^{o} / @ 90^{o}			
4.	Lateral ➢ medial @ 180^{o} / @ 90^{o}			
5.	Internal rotation			
6.	External rotation			
7.	Anterior ➢ posterior			
8.	Posterior ➢ anterior			
9.	Quadrant (scouring) test			
SPECIAL TESTS			Right	Left
1.	Patrick FABER Test			
2.	Trendelenberg Test			
3.	Craig's Test			
4.	Leg Length	Actual		
		Apparent		
5.	Sign of the Buttock			
6.	Thomas Test (hip flexion contracture)			
7.	Rectus Femoris Contracture Test			
8.	Iliopsoas contracture Test			
9.	Ely's Test (rectus femoris hypertonicity)			
10.	Ober's Test (ITB contracture)			
11.	Noble Compression Test (ITB Friction Syndrome)			
12.	Piriformis Test			
13.	Hamstrings	Hamstring Contracture Test		
		Tripod Test		

APPENDIX F

KNEE REGIONAL EXAMINATION

Patient:_____ File:_____ Date:_____
Intern:_____ Signature:_____
Clinician:_____ Signature:_____

! **OBSERVATION (**Standing, Seated and during gait cycle).

A. Anterior view
Genu Varum:_____
Genu Valgum:_____
Patellar position: _____
Tibial Torsion: _____
Skin:_____
Swelling:_____

B. Lateral view
Genu Recurvatum:_____
Patella Alta:_____
Patella Baja:_____
Skin:_____

C. Posterior view
Swelling:_____
Skin:_____

D. General
Movement symmetry:_____
Structures symmetry:_____

! **ACTIVE MOVEMENTS**
Flexion (0 - 135E)_____
Extension (0 - 15E)_____
Medial Rotation (20 - 30E)_____
Lateral rotation (30 - 40E)_____
Patellar movement_____

! **PASSIVE MOVEMENTS**
Tissue approx_____
Bone-bone_____
Tissue stretch_____
Tissue stretch_____

! **RESISTED ISOMETRIC MOVEMENTS**
Knee: Flexion:_____
Extension:_____
Internal rotation:_____
External rotation:_____

Ankle: Plantarflexion_____
Dorsiflexion_____

! **LIGAMENTOUS ASSESSMENT**

One-Plane Medial Instability
Valgus stress (abduction)
Extended_____
Resting Position_____

One-Plane Lateral Instability
Varus stress (adduction)
Extended_____
Resting Position_____

One-Plane Anterior Instability
Lachman Test (0-30°)_____
Anterior Drawer Sign _____

One-Plane Posterior Instability
Posterior "sag" Sign_____
Posterior Drawer Test_____

Anterolateral Rotatory Instability
Slocum Test_____
Macintosh Test_____

Anteromedial Rotatory Instability
Slocum Test_____

Posterolateral Rotatory Instability
Jacob _____
Hughston's Drawer Sign_____
Reverse pivot shift test_____

Posteromedial Rotatory Instability
Hughston's Drawer Sign_____

! TESTS FOR MENISCUS INJURY

McMurray_____ Anderson med-lat grind_____
"Bounce Home"_____ Apley's_____

! PLICA TESTS

Mediopatellar_____ Plica Hughston's Plica_____
Plica "Stutter"_____

! TESTS FOR SWELLING

Brush/Stroke Test_____ Patellar Tap Test_____

! TESTS FOR PATELLA FEMORAL PAIN SYNDROME

Clarke's Sign_____ Passive patella tilt test_____
Waldron test_____

! OTHER TESTS

Wilson's_____ Quadriceps Contusion Test_____
Fairbank's_____ Leg Length Discrepancy_____
Noble Compression_____

! JOINT PLAY

Movement of the tibia on the femur P | A:_____ A | P:_____
Translation of the tibia on the femur M | L:_____ L | M:_____
Long axis distraction of the tibiofemoral joint _____
Inf, sup, lat, + med glide of the patella _____
Movement of the inf. tibiofibular joint A | P:_____ P | A:_____
Movement of the sup. tibiofibular joint A | P:_____ P | A:_____
Movement of the sup. tibiofibular joint S | I :_____ I | S_____

! PALPATION

Tenderness Swelling
Joint line Nodules/exostoses
Ligaments Muscles: thigh:
Patella: Leg :
Patella tendon: Popliteal artery:
Bursae:

! REFLEXES AND CUTANEOUS DISTRIBUTION

	R	L
Patellar Reflex (L3,L4)		
Medial Hamstring Reflex (L5,S1)		

! DERMATOMES

	R	L		R	L
L2			S1		
L3			S2		
L4			S3		
L5					

DURBAN UNIVERSITY of TECHNOLOGY
Making Knowledge Useful

APPENDIX G

Foot and ankle regional examination

Patient:_____ File no:_____ Date:_____
Intern / Resident:_____ Signature:_____
Clinician:_____ Signature:_____

Observation
Gait analysis (antalgic limp,toe off, arch, foot alignment, tibial alignment).

_____
Swelling_____
Heloma dura / molle_____
Skin_____
Nails_____
Shoes_____
Contours (achilles tendon, bony prominences)_____

Active movements

Weight bearing:	**R**	**L**	***Non weight bearing:***	**R**	**L**
Plantar flexion			50°		
Dorsiflexion			20°		
Supination					
Pronation					
Toe dorsiflexion			40°(mtp)		
Toe plantar flexion			40° (mtp)		
		Big toe dorsiflexion (mtp) (65-70°)			
		Big toe plantar flexion (mtp) 45°			
		Toe abduction + adduction			
		5° first ray dorsiflexion			
		5° first ray plantar flexion			

Passive movement motion palpation (Passive ROM quality, ROM overpressure, joint play)

	R	**L**		**R**	**L**
Ankle joint: *Plantarflexion*			Subtalar joint: *Varus*		
Dorsiflexion			*Valgus*		
Talocrural: *Long axis distraction*			Midtarsal:*A-P glide*		
First ray: *Dorsiflexion*			*P-A glide*		
Plantarflexion			*rotation*		
Circumduction of forefoot on fixed rearfoot			Intermetatarsal glide		
			Tarso metatarsal joints: *A-P*		
Interphalangeal joints: *L A dist*			Metatarsophalangeal dorsiflexion (with associated plantar flexion of each toe		
A-P glide					
lat and med glide					
rotation					

Resisted Isometric movements

	R	L		R	L
Knee flexion			Pronation (eversion)		
Plantar flexion			Toe extension (dorsiflexion)		
Dorsiflexion			Toe flexion (plantar flexion)		
Supination (inversion)					

Neurological	**R**	**L**
Dermatomes		
Myotomes		
Reflexes		
Balance/proprioception		

Special tests	**R**	**L**
Anterior drawer test		
Talar tilt		
Thompson test		
Homan sign		
Tinel's sign		
Test for rigid/flexible flatfoot		
Kleiger test (med. deltoid)		

Alignment	**R**	**L**
Heel to ground		
Feiss line		
Tibial torsion		
Heel to leg (subtalar neutral)		
Subtalar neutral position:		
Forefoot to heel (subtalar & Midtarsal neutral)		
First ray alignment		
Digital deformities		
Digital deformity flexible		

Palpation

Anteriorly	**R**	**L**
Medial maleoli		
Med tarsal bones, tibial (post) artery		
Lat.malleolous, calcaneus, sinus tarsi, and cuboid bones		
Inferior tib/fib joint, tibia, mm of leg		
Anterior tibia, neck of talus, dorsalis pedis artery		

Posteriorly

Calcaneus, Achilles tendon, Musculotendinous junction		

Plantarily

Plantar muscles and fascia		
Sesamoids		

APPENDIX H

DURBAN UNIVERSITY OF TECHNOLOGY

Patient Name: ***File #:*** ***Page:***

Date: ***Visit:*** ***Intern:***
Attending Clinician: ***Signature:***

S: ***Numerical Pain Rating Scale (Patient)*** ***Intern Rating*** ***A:***
Least 0 1 2 3 4 5 6 7 8 9 10 Worst ☐

0: ***P:***

E:

Special attention to: ***Next appointment:***

Date: ***Visit:*** ***Intern:***
Attending Clinician: ***Signature:***

S: ***Numerical Pain Rating Scale (Patient)*** ***Intern Rating*** ***A:***
Least 0 1 2 3 4 5 6 7 8 9 10 Worst ☐

O: ***P:***

E:

Special attention to: ***Next appointment:***

Date: ***Visit:*** ***Intern:***
Attending Clinician: ***Signature***

S: ***Numerical Pain Rating Scale (Patient)*** ***Intern Rating*** ***A:***
Least 0 1 2 3 4 5 6 7 8 9 10 Worst ☐

O: ***P:***

E:

Special attention to: ***Next appointment:***

APPENDIX I

LETTER OF INFORMATION AND INFORMED CONSENT

Dear Patient

Welcome to my research.

Title of research: The relative effectiveness of three full kinetic chain treatment protocols for osteoarthritis of the knee: Manual therapy, rehabilitation and a combination thereof.

NAME OF RESEARCH STUDENT
Lauren Dwyer Contact number: 083 501 4897

NAME OF RESEARCH SUPERVISOR
Dr Charmaine Korporaal (M.Tech: Chiropractic, CCFC, CCSP, ICSSD)
Contact number: 031 373 2611

NAME OF RESEARCH CO-SUPERVISOR
Dr James Brantingham (DC, PhD; Director of Research and Associate Professor
Cleveland Chiropractic College Los Angeles}
Contact email address: james.brantingham@cleveland.edu

The aim of this study is to evaluate the effectiveness of three full kinetic chain treatment protocols in the management of osteoarthritis of the knee.

Research procedure: At least sixty people will be required to complete this study. These participants will be randomly divided into three treatment groups of twenty patients each. All patients will receive treatment that has been proven effective in various studies; however, the specific treatment you receive will depend on the group you are assigned to.

At the initial consultation, a full case history, physical examination and various regional examinations will be performed. You will receive your first treatment and be briefed on what is required of you throughout the study. Thereafter, you will be required to attend 5 further consultations over a 3 week period followed by a 7th consultation in the 4th week in order to take the final measurements. At certain consultations one or more of the following will occur:

- You will be asked to complete questionnaires pertaining to your knee pain
- An Inclinometer will be used to measure range of motion of your knee joint
- X-rays of the knee and/or other joints of the lower limb will need to be taken in order to make a diagnosis.

Depending on which group you fall into, you may also be required to fill in a diary to keep track of whether you have performed your daily exercises and what medication you may have taken on a particular day and why (e.g. took an anti-inflammatory because of increased swelling).

Patient medication and lifestyle: You will be permitted to continue taking any medication that you have started more than 6 weeks before joining the research. Any change in dosage must be noted in the diary provided. If you start taking any new medication, or undergo any other form of treatment for your knee, you may be excluded from the study.

Please try not to alter your normal lifestyle or daily activities in any way as this could interfere with the results of this study.

Benefits of taking part in the research: The allocated treatments, in most cases, decrease the pain, stiffness and dysfunction in the region of the knee joint.

Risks of taking part in the research: The treatment is safe and is unlikely to cause any discomfort or adverse side effects, although post treatment stiffness has been noted in some circumstances.

Costs: All treatments will be performed under the supervision of a qualified Chiropractor and will be free of charge.

Confidentiality: The results of the study will be made available in the Durban University of Technology library in the form of a mini-dissertation. If you consent, the results of this study will also be used in a larger study which is being conducted in conjunction with Cleveland Chiropractic College Las Angeles, an American chiropractic college and research institute. Every measure will be taken to ensure that all patient information remain strictly confidential.

You are free to withdraw from this study at any stage. However, if you choose to withdraw, you may be asked to return for the allocated data collection sessions so that information can be gathered for statistical purposes.

Please feel free to ask any questions regarding any aspect of this study. Your full co-operation will assist the Chiropractic profession in expanding its knowledge of this condition.

Persons to Contact in the Event of Any Problems or Queries:

Supervisor: Dr.C.Korporaal, 0313732611(w), charmak@dut.ac.za
Co-supervisor: Dr J. Brantinghamjames.brantingham@cleveland.edu
HoD: Dr C, Korporaal, 0313732611(w), charmak@dut.ac.za
Faculty Officer: Vikesh Singh, 0313732701 (w), Faculty of Health offices.

Statement of Agreement to Participate in the Research Study:

I,…… (subject's full name),
…….(ID number), have read this document in its entirety and understand its contents. Where I have had any questions or queries, these have been explained to me by
…….. to my satisfaction.

Furthermore, I fully understand that I may withdraw from this study at any stage without any adverse consequences and my future health care will not be compromised. I, therefore, voluntarily agree to participate in this study.

Subject's name (print): …….. ……..
Subject's signature :…….. Date:……

Researcher's name (print): ……... ……
Researcher's signature:……... ……Date:.....

Witness name (print) : …….....
Witness signature: …….....Date:…….

Thank-you
Yours Faithfully,

Lauren Dwyer
(Chiropractic Intern)

Dr Charmaine Korporaal
(Supervisor)

APPENDIX J

WESTERN ONTARIO AND McMASTER UNIVERSITIES OSTEOARTHRITIS INDEX (WOMAC)

Name:_____

Date:___/___/___ Visit Number:___

Section A

The following questions concern the amount of **PAIN**you are currently experiencing in your knee. For each situation please enter the amount of pain recently experienced. (Please mark your answers with a straight vertical {up-and-down} mark on the line).

1. Walking on a flat surface

NO PAIN |_____| EXTREME PAIN

2. Going up or down stairs

NO PAIN |_____| EXTREME PAIN

3. At night while in bed

NO PAIN |_____| EXTREME PAIN

4. Sitting or lying

NO PAIN |_____| EXTREME PAIN

5. Standing upright

NO PAIN |_____| EXTREME PAIN

Section B

The following questions concern the amount of **JOINT STIFFNESS**(not pain) you are currently experiencing in your knee. Stiffness is a sensation of restriction or slowness in the case with which you move your joints.

1. How severe is your stiffness after first wakening in the morning?

NO STIFFNESS |_____| EXTREME STIFFNESS

2. How severe is your stiffness after sitting, lying or resting later in the day?

NO STIFFNESS |_____| EXTREME STIFFNESS

Section C

Question: What **DEGREE OF DIFFICULTY** do you have with:

1. Descending stairs.

NO DIFFICULTY ——— EXTREME DIFFICULTY

2. Ascending stairs

NO DIFFICULTY ——— EXTREME DIFFICULTY

3. Rising from sitting

NO DIFFICULTY ——— EXTREME DIFFICULTY

4. Standing

NO DIFFICULTY ——— EXTREME DIFFICULTY

5. Bending to floor

NO DIFFICULTY ——— EXTREME DIFFICULTY

6. Walking on a flat surface

NO DIFFICULTY ——— EXTREME DIFFICULTY

7. Getting in/out of car

NO DIFFICULTY ——— EXTREME DIFFICULTY

8. Going shopping

NO DIFFICULTY ——— EXTREME DIFFICULTY

9. Putting on socks/stockings

NO DIFFICULTY ——— EXTREME DIFFICULTY

10. Rising from bed

NO DIFFICULTY — EXTREME DIFFICULTY

11. Taking off socks/stockings

NO DIFFICULTY — EXTREME DIFFICULTY

12. Lying in bed

NO DIFFICULTY — EXTREME DIFFICULTY

13. Getting in/out of bath

NO DIFFICULTY — EXTREME DIFFICULTY

14. Sitting

NO DIFFICULTY — EXTREME DIFFICULTY

15. Getting on/off toilet

NO DIFFICULTY — EXTREME DIFFICULTY

16. Heavy domestic duties

NO DIFFICULTY — EXTREME DIFFICULTY

17. Light domestic duties

NO DIFFICULTY — EXTREME DIFFICULTY

APPENDIX K

McMASTER OVERALL THERAPY EFFECTIVENESS (OTE) TOOL

Patient No.□□□□□□ Visit No.□□

Question 1: Overall Treatment Evaluation
Since treatment started, has there been any change in your ACTIVITY LIMITATION, SYMPTOMS AND/OR FEELINGS related to your knee osteoarthritis?

Please indicate if there has been any change by checking **ONE** of the three boxes below (Better/About the Same/Worse):

□ Better □About the Same □ Worse
⇓ ⇓
If you have checked **ABOUT THE SAME**,
⇓ **Please stop here**. ⇓

If you have checked the box BETTER:

How much BETTER would you say your ACTIVITY LIMITATION, SYMPTOMS AND/OR FEELINGS have been since treatment started?

Please choose ONE of the options below:

□ Almost the same, hardly better at all
□ A little better
□Somewhat better
□Moderately better
□ A good deal better
□ A great deal better
□A very great deal better

If you have checked the box WORSE:

How much WORSE would you say your ACTIVITY LIMITATION, SYMPTOMS AND/OR FEELINGS Have been since treatment started?

Please choose ONE of the options below:

□ Almost the same, hardly worse at all
□ A little worse
□ Somewhat worse
□ Moderately worse
□ A good deal worse
□ A great deal worse
□ A very great deal worse

Question 2: Overall Treatment Effect

Answer the following question whether or not you answered BETTER or WORSE and what your response was. Note if you have improved, the change will be important since you likely will be able to carry out your responsibilities with greater ease and comfort compared to before the study. If on the other hand you are worse, then you will have more difficulty carrying out your responsibilities; this will also be important for you as you have more difficulty with your activities.

Is this change (BETTER/WORSE) important to you in carrying out your daily activities?

□Not important
□Slightly important
□Somewhat important
□Moderately important
□ Important
□ Very important
□Extremely important

APPENDIX L

BECK DEPRESSION INVENTORY

1. SADNESS	**2. PESSIMISM**
0 I do not feel sad **1** I feel sad much of the time **2** I am sad all the time **3** I am so sad/unhappy that I can't stand it	**0** I am not discouraged about my future **1** I feel more discouraged about my future than I used to be **2** I do not expect things to work out for me **3** I feel my future is hopeless and will only get worse
3. PAST FAILURE	4. **LOSS OF PLEASURE**
0 I do not feel like a failure **1** I have failed more than I should have **2** As I look back, I see a lot of failure **3** I feel I am a total failure as a person	**0** I get as much pleasure as I ever did from the things I enjoy **1** I don't enjoy things as much as I used to **2** I get very little pleasure from the things I used to enjoy **3** I can't get any pleasure from the things I used to enjoy
5. GUILTY FEELINGS	**6. PUNISHMENT FEELINGS**
0 I don't feel particularly guilty **1** I feel guilty over things I have done or should have done **2** I feel guilty most of the time **3** I feel guilty all of the time	**0** I don't feel I am being punished **1** I feel I may be punished **2** I expect to be punished **3** I feel I am being punished
7. SELF-DISLIKE	**8. SELF-CRITICALNESS**
0 I am disappointed in myself **1** I dislike myself **2** I feel the same about myself as ever **3** I have lost confidence in myself	**0** I don't criticise or blame myself more than usual **1** I am more critical of myself than I used to be **2** I criticise myself for all my faults **3** I blame myself for everything bad that happens
9. SUICIDAL THOUGHTS OR WISHES	**10. CRYING**
0 I don't have any thoughts of killing myself **1** I have thoughts of killing myself, but would not carry them out **2** I would like to kill myself **3** I would kill myself if I had a chance	**0** I don't cry any more than I used to **1** I cry more than I used to **2** I cry over every little thing **3** I feel like crying, but I can't
11. AGITATION	**12. LOSS OF INTERESTS**
0 I am no more restless or wound up than usual **1** I feel more restless or wound up than usual **2** I am so restless or agitated that it's hard to stay still **3** I am so restless or agitated that I have to keep moving or doing something	**0** I have not lost interest in other people or activities **1** I am less interested in other people or things than before **2** I have lost most of my interest in other people or things **3** It's hard to get interested in anything
13. INDECISIVENESS	**14. WORTHLESSNESS**
0 I make decisions about as well as ever **1** I find it more difficult to make decisions than usual **2** I have much greater difficulty in making decisions than I used to **3** I have trouble making any decisions	**0** I do not feel I am worthless **1** I don't consider myself as worthwhile and useful as I used to **2** I feel more worthless as compares to other people **3** I feel utterly worthless
15. CHANGE IN APPETITE	**16. CHANGES IN SLEEP PATTERN**
0 I have not experienced any change in appetite **1a** My appetite is somewhat less than usual **1b** My appetite is somewhat greater than usual **2a** My appetite is much less than before **2b** My appetite is much greater than before **3a** I have no appetite **3b** I crave food all the time	**0** I have not experienced any change in my sleeping pattern **1a** I sleep somewhat more than usual **1b** I sleep somewhat less than usual **2a** I sleep a lot more than usual **2b** I sleep a lot less than usual **3a** I sleep most of the day **3b** I wake up 1-2 hours earlier and can't get back to sleep
17. IRRITABILITY	**18. LOSS OF ENERGY**
0 I am no more irritable than usual **1** I am more irritable than usual **2** I am much more irritable than usual **3** I am irritable all the time	**0** I have as much energy as ever **1** I have less energy than I used to have **2** I don't have enough energy to do very much **3** I don't have enough energy to do anything
19. CONCENTRATION DIFFICULTY	**20. TIREDNESS OR FATIGUE**
0 I can concentrate as well as ever **1** I can't concentrate as well as usual **2** It's hard to keep my mind on anything for very long **3** I find I can't concentrate on anything	**0** I am no more tired or fatigued than usual **1** I get more tired or fatigued more easily than usual **2** I am too tired or fatigued to do lots of the things I used to do **3** I am too tired or fatigued to do most of the things I used to do
21. LOSS OF INTEREST IN SEX	
0 I have not noticed any recent change in my interest in sex **1** I am less interested in sex than I used to be **2** I am much less interested in sex now **3** I have lost interest in sex completely	**TOTAL = _____**

APPENDIX M
BERG BALANCE SCALE

1. SITTING TO STANDING		**8. REACHING FORWARD WITH OUTSTRETCHED ARM**	
Able to stand without using hand and stabilise indep	4	Can reach forward confidently > 25 cm	4
Able to stand independently using hands	3	Can reach forward >12 cm safely	3
Able to stand using hands after several tries	2	Can reach forward >5cm safely	2
Needs minimal aid to stand or stabilise	1	Reaches forward but needs supervision	1
Needs moderate or maximal assist to stand	0	Loses balance while trying; requires external support	0
2. STANDING UNSUPPORTED		**9. PICK UP OBJECT FROM FLOOR**	
Able to stand safely for two minutes	4	Able to pick up slipper safely and easily	4
Able to stand two minutes with supervision	3	Able to pick up slipper but needs supervision	3
Able to stand 30 seconds unsupported	2	Unable to pick up but reaches to 2-5cm from slipper and keeps balance independently	2
Needs several tries to stand 30 seconds unsupported	1	Unable to pick up and needs supervision while trying	1
Unable to stand 30 seconds unassisted	0	Unable to try /needs assist to keep from losing balance or falling	0
3. SITING UNSUPPORRTED		**10.TURN TO LOOK BEHIND**	
Able to sit safely and securely 2 minutes	4	Looks behind from both sides and weight shifted well	4
Able to sit two minutes under supervision	3	Looks behind one side only other side shows left weight shift	3
Able to sit 30 seconds	2	Turns sideways only but maintains balance	2
Able to sit 10 seconds	1	Needs supervision when turning	1
Unable to sit without support 10 seconds	0	Needs assist to keep from losing balance or falling	0
4.STANDING TO SITTING		**11. TURN 360 º**	
Sits safely with minimal use of hands	4	Able to turn 360 degrees safely in 4 seconds or less	4
Controls descent by using hands	3	Able to turn 360degrees safely one side only in 4 seconds or less	3
Uses back of legs against chair to control descent	2	Able to turn 360 degrees safely but slowly	2
Sits independently but has uncontrolled descent	1	Needs close supervision or verbal cueing	1
Needs assistance to sit	0	Needs assistance while turning	0
5.TRANSFERS		**12. PLACE ALTERNATRE FOOT ON STEP OF STOOL**	
Able to transfer safely with minor use of hands	4	Abel to stand independently and safely and complete 8 steps in 20 seconds	4
Able to transfer safely minor use of hands	3	Able to stand independently and compete 8 steps > 20 seconds	3
Able to transfer with verbal cueing and/or supervision	2	Able to complete 4 steps without aid with supervision	2
Needs one person to assist	1	Able to complete > 2 steps needs minimal assist	1
Needs two people to assist	0	Needs assistance to keep from falling / unable to try	0
6. STANDING UNSUPPORTED WITH EYES CLOSED		**13.TANDEM STANCE**	
Able to stand 10 seconds safely	4	Able to place foot tandem independently and hold 30 seconds	4
Able to stand 10 seconds with supervision	3	Able to place foot ahead of other independently and hold 30 seconds	3
Able to stand 3 seconds	2	Able to take small step independently and hold 30 seconds	2
Unable to keep eyes closed 3 seconds but stays steady	1	Needs help to step but can hold 15 seconds	1
Needs help to keep from falling	0	Loses balancer whilst stepping or standing	0
7.STANDING UNSUPPORTED WITH FEET TOGETHER		**14. STANDING ON ONE LEG**	
Able to place feet together independently and stand one minute safely	4	Able to lift le independently and hold > 10 seconds	4
Able to pace feet together independently and stand one minute with supervision	3	Able to lift leg independently and hold 5-10 seconds	3
Able to place feet together independently but unable to hold for 30 seconds	2	Able to lift leg independently and hold = or > 3 seconds	2
Needs help to attain position but unable to stand 15 seconds feet together	1	Tries to lift leg unable to hold 3 seconds but remains standing independently	1
Needs help to attain position and unable to hold for 15 seconds	0	Unable to try or needs assist to prevent fall	0

APPENDIX N

RANGE OF MOTION

	Previsit 1	Previsit 4	Visit 7
Date			
Flexion			
Extension			

APPENDIX O

MEDICATION AND EXERCISE DIARY

Medication Taken:

	Day	Week 1	Week 2	Week 3	Week 4
Type, Amount, and Reason (e.g. Ibuprofen x 2; pain and swelling)	1.				
	2.				
	3.				
	4.				
	5.				
	6.				
	7.				

Strengthening Exercises:

		Week 1	Week 2	Week 3	Week 4
1. **AND**	Quad setting (daily)				
2. **AND**	Standing knee extensions (3 x per week)				
3a. **OR**	Seated leg press (3 x per week)				
3b. **OR**	Partial squat (3 x per week)				
3c.	Step-ups (3 x per week)				

Stretch Exercises:

	Week 1	Week 2	Week 3	Week 4
1. Standing calf				
2. Hamstring				
3. Quadriceps				
4. Iliopsoas				
5. Adductor and Gracilis				
6. TFL and Glut med				

Range of Motion Exercises:

	Week 1	Week 2	Week 3	Week 4
Knee in mid-extension to full extension				
Knee in mid-flexion to full flexion				

Walking

	Week 1	Week 2	Week 3	Week 4
Walking on a treadmill or flat surface (duration and distance covered)				

APPENDIX P

EXERCISE BROCHURE

Deyle Protocol

Stretches
Both sides should be stretched

Calf Stretch 3 reps of 30 second holds

- Stand with your palms on a wall, with your leg in back, heel on floor and lean hips into wall to increase the stretch
- Maintain a neutral spine posture

Hamstring Stretch 3 reps of 30 second holds

- Lying on the back with the hip flexed the leg should be up in the air
- Use a strap around the back of the calf to pull the leg forward pulling until a gentle stretch is felt

Quadriceps Stretches *- 3 versions-* 3 reps of 30 second holds

Standing

- Standing draw one knee to your chest
- Grasp the ankle with the ipsilateral hand
- Tuck your butt – posterior pelvic tilt – and bring your ankle back until you have slight leg extension or as far as you need to feel a slight tug
- Maintain the posterior pelvic tilt
- To increase the stretch bring the involved knee closer to the stance knee

Standing with a chair

- Stand to the side of a chair with your hand on the back of it for support
- Place the knee of the leg you are stretching on the seat of the chair, your stance leg should be forward of the chair a stride length
- Push your hips forward making sure to maintain a posterior pelvic tilt
- This method will stretch the hip flexors as well

Side lying

- Lie on your left side with the left arm extended and head resting on it.
- Tuck the butt under as in a posterior pelvic tilt.
- Bring the right ankle back towards the glutes.
- Reach back with the right hand and gently hold the ankle keeping the right knee parallel to the floor.
- Push hips forward and slightly extend the hip until you feel a gentle stretch to the hip flexors

Range of Motion Exercises

The next two exercises are performed in "long sitting." Long sitting is performed with the patient seated with their legs extended straight out in front of them. For patient comfort you can have them sit with a wall behind them for support with or without a pillow. You could also have them sit with a slanted cushion under them for low back support.

Perform 2 sets of 30 seconds bouts with a 3 second hold at end range. Do this for each exercise below

Knee in mid flexion bring to full extension
Knee in mid flexion bring to full flexion

Stationary Bike Start at 5 minutes and build the amount of time as they are able.

Resistive Exercises

Quad Setting Each contraction should be held for 6 seconds. Perform 10 repetitions with a 10 second rest between sets.

- The patient is in the long sitting position on a mat on the floor or on a table with the involved leg extended
- The uninvolved leg can be flexed or extended depending on patient comfort
- Have the patient perform an isometric contract of the quadriceps muscle on the involved side
- This contraction should be sufficient to cause the patella to move proximally

Closed Kinetic Chain Progressions

Choose **one** of the exercises from the exercises listed below. Choose the one that is pain-free.

Standing Terminal Knee Extensions One 30 second bout.

- Patient standing with a Theraband around the knee tied to the leg of a table or on a door handle
- The knee should be in a partially flexed position
- Actively move the knee into extension against the resistance of the Theraband
- Gradually increase resistance by shortening the Theraband or standing farther away from the point where the Theraband is fixed.

Seated Leg Press One 30 second bout for each leg

- Patient is seated in a chair with one knee in flexion, the other foot is on the floor for stability
- The Theraband is wrapped under the involved foot while the patient holds the ends near their trunk
- The patient then extends their leg to 0°, slowly bring the leg back to flexion
- After the 30 second bout switch legs and do the same on the other side

** Increase resistance as patient gains strength. Resistance can be added by shortening the Theraband or adding another Theraband

Double Knee Dip One 30 second bout – perform 1 second on the concentric/2 seconds on the eccentric

- Patient stands with both feet on a Theraband with the ends in their hands at about waist level
- Stand with the knees slightly flexed and the great toe pointed very slightly out
- The patient is instructed to slowly lower themselves ONLY a few inches to start with
- Return to the slight flexed knee and repeat
- As the patient improves in strength and form they can increase the amount of flexion and eventually move to one leg (see next page for one legged dips)

** If the patient is unstable, frail or elderly put a stable chair (or a bar) in front of them to hold on to and don't use the Theraband

**The knees should remain pointed outwards slightly through the whole excursion

** The hinge points are the hips, knees and ankles maintaining a straight back

Single Knee Dip One 30 second bout – perform 1 second on the concentric/2 seconds eccentric

- The patient should place a chair next to them on the uninvolved side with their hand placed on the back of the chair for balance until they have the coordination to do it without the assistance
- Stand on one leg only using the same form as the double knee dip, slowly lowering a few inches, returning to slight flexion
- Increase the depth of the dip as the patients functional range and strength improve

Step Ups One 30 second bout

- The patient can use stairs or a step up exerciser with risers like the "Buns of Steel" step
- To start the step should be no higher than 4"
- Begin by having the patient face the step or riser
- Start by placing the right foot in the middle of step and step up
- Then bring the left leg on to the step as well
- Step down with their left leg first and then continue on down with their right leg
- Increase the height as your patient gains strength
- Reverse the starting leg every other day or half way through the exercise session

APPENDIX Q

RANDOMISED ALLOCATION CHART

STUDY A (1st Stratification ages **38-59**) Groups 1, 2, and 3

0001: Group 1	0002: Group 3	0003: Group 2	0004: Group 2	0005: Group 3	0006: Group 1
0007: Group 2	0008: Group 3	0009: Group 2	0010: Group 1	0011: Group 3	0012: Group 1
0013: Group 1	0014: Group 3	0015: Group 2	0016: Group 1	0017: Group 2	0018: Group 3
0019: Group 3	0020: Group 2	0021: Group 2	0022: Group 1	0023: Group 1	0024: Group 3
0025: Group 2	0026: Group 1	0027: Group 1	0028: Group 3	0029: Group 2	0030: Group 3

STUDY B (2nd Stratification ages **60-80**) Groups 1, 2, and 3

0001: Group 3	0002: Group 2	0003: Group 1	0004: Group 1	0005: Group 2	0006: Group 3
0007: Group 1	0008: Group 3	0009: Group 3	0010: Group 2	0011: Group 1	0012: Group 2
0013: Group 3	0014: Group 1	0015: Group 2	0016: Group 1	0017: Group 2	0018: Group 3
0019: Group 1	0020: Group 3	0021: Group 2	0022: Group 2	0023: Group 3	0024: Group 1
0025: Group 3	0026: Group 1	0027: Group 2	0028: Group 2	0029: Group 1	0030: Group 3

More when needed (for ages **60-80**):

0031: Group 1	0032: Group 2	0033: Group 3	0034: Group 1	0035: Group 2	0036: Group 3
0037: Group 2	0038: Group 3	0039: Group 1	0040: Group 3	0041: Group 1	0042: Group 2
0043: Group 2	0044: Group 1	0045: Group 3	0046: Group 3	0047: Group 1	0048: Group 2

APPENDIX R
ETHICS CLEARANCE CERTIFICATE

DURBAN UNIVERSITY of TECHNOLOGY

Faculty of Health Sciences

ETHICS CLEARANCE CERTIFICATE

Student Name	Lauren Dwyer	**Student No**	20600346
Ethics Reference Number	31/10	**Date of FRC Approval**	30/06/2010
Qualification	M.Tech: Chiropractic		
Research Title:	The relative effectiveness of three full kinetic chain treatment protocols for osteoarthritis of the knee: Manual therapy, rehabilitation and a combination thereof.		

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	✓		

[redacted]
SIGNATURE OF STUDENT/RESEARCHER — 21/9/2010 DATE

[redacted]
SIGNATURE OF SUPERVISOR/S — 21/9/2010 DATE

[redacted]
SIGNATURE OF HEAD OF DEPARTMENT — 22/9/2010 DATE

[redacted]
SIGNATURE: CHAIRPERSON OF RESEARCH ETHICS COMMITTEE — 20/10/2010 DATE