

The effect of three manipulative treatment protocols on quadriceps muscle strength in patients with Patellofemoral Pain Syndrome.

By

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This dissertation is submitted in partial compliance with the requirements for the Master's Degree in Technology:

Chiropractic

In the Faculty of Health at the Durban Institute of Technology.

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DEDICATION

This thesis is dedicated to my late grandfather, Hans-George Hillermann, who passed away on 5 June 2003. Opa- Danke für alles was du mir gelehrt hast. Ich freue mich auf das wieder sehen!

AKNOWLEDGEMENTS

Firstly I would like to thank GOD for always being there for me and for putting me on this path. Make me phenomenal to you.

To Mom and Dad. Thank you for all your support! You both mean the world to me.

To my brother Hans. Thank you for always pulling me out of bed so early in the mornings and dragging me down to the beach. Those surfing trips formed the base for a great friendship and our truly unique relationship. I'm just sorry that I had to whip your butt once we got into the water! ☺ Thank you.

To my sister Gisela. You are the rose amongst the thorns! Your cheerful attitude towards life has always been a breath of fresh air in mine. Thanx Sista.

To Gareth. Thank you for all the lifts to Tech and years of laughter and fun. I look forward to all the things we are going to get up to in the future.

To Dr. C. Korporaal. I don't think any student or future student at DIT can ever thank you enough for what you do for the profession and us. Thank you for always going the extra mile. You're a legend!

To Dr. N. Gomes. Thank you for your guidance and effort during the research process. All the best!

To Dennis Jackson. It was great working with you on this study. Thank you very much.

To Mr. Brooks and his team at the KwaZulu-Natal Department of Health. Thank you for the bursary that kept me going for the last six years.

To Linda and Pat. Thank you for all your effort in the clinic. You guys run a smooth ship.

A big THANK YOU has to go out to all the patients who participated in this study. Without you this would not have been possible.

Lastly, to the Grant, Craig and Marlon from the caffeine high 5. Without you guys I would not have made it through the course. Thank you!

ABSTRACT

Knee joint pathologies, in general, are associated with a loss of knee-extensor muscle strength. This weakness has been attributed to arthrogenic muscle inhibition (AMI). Manipulation of the sacroiliac (SI) has been shown to significantly reduce AMI and increase the strength in the quadriceps muscle group. Although both the knee and SI joints have been linked to AMI of the quadriceps muscle group, no studies have been conducted showing that manipulating the tibio-femoral (knee) joint has any effect on quadriceps muscle strength or AMI.

The purpose of this study was therefore to verify whether manipulation of the knee (tibio-femoral) joint is as effective as SI joint manipulation in increasing quadriceps muscle strength in PFPS patients. This study also investigated the effect of combining manipulative therapy of the tibio-femoral and SI joints on quadriceps muscle strength in PFPS patients.

This pilot study was a prospective clinical trial. Thirty subjects suffering from PFPS were evaluated in an experimental fashion, using standardized isokinetic testing protocols. At the first consultation, subjects underwent a full case history, physical, lumbar spine, and knee regional examinations.

Using convenience, purposive sampling, the thirty subjects were allocated into three treatment groups. Subjects in group A received a long axis distraction manipulation of the tibio-femoral joint; subjects in group B received a sacroiliac joint manipulation on the ipsilateral side of the knee pain; and subjects in group C received both a long axis distraction manipulation of the tibio-femoral joint and a sacroiliac joint manipulation on the ipsilateral side of the knee pain. Subjects who suffered from bilateral PFPS were asked to make a subjective judgment as to which knee was worst. Only that side was then tested.

The second consultation took place at a rehabilitation centre where concentric-concentric isokinetic testing was performed by a biokineticist, using the Cybex Orthotron II Isokinetic Rehabilitation System. Immediately after baseline measurements of quadriceps and hamstring muscle strengths were taken, the subjects received a treatment consisting of one of three manipulative treatment protocols. The subjective awareness of pain was also measured using the Numerical Pain Rating Scale (NPRS), pre and post treatment.

Descriptive statistics were performed using frequency distribution tables and graphs. Descriptions on the means and standard deviations were also performed. Non-parametric testing was used for statistical analyses of the data. Intra-group comparison consisted of the Wilcoxon Signed Ranks test for matched pairs, which tested both the subjective and objective data for a significant change as a result of the intervention. Inter-group comparison consisted of the Kruskal-Wallis H test, which was used to test for a significant difference between the treatment groups for all variables tested. Finally, the Pearson Product Moment Correlation coefficient was applied to test for a significant relationship between the level of perceived pain and the maximum voluntary quadriceps or hamstring forces.

The results showed that an improvement in both the maximum voluntary quadriceps and hamstring forces occurred immediately after manipulation of the tibio-femoral joint, the sacroiliac joint and a combination of adjustments of the tibio-femoral and sacroiliac joints.

With regards to the quadriceps force, a significant improvement occurred only after a sacroiliac manipulation on the ipsilateral side of the knee pain was administered. The hamstring force however, improved significantly after both a sacroiliac joint manipulation and a combination of the sacroiliac and tibio-femoral joint manipulations.

The study failed to show any significant difference between the three manipulative treatment protocols, in terms of their effect on quadriceps and

hamstring strengths. There was no significant correlation between the level of perceived pain and the maximum voluntary quadriceps or hamstring forces.

Although this study showed some significant changes in the quadriceps and hamstring strengths following manipulation, no definitive conclusions could be drawn as to which manipulative treatment protocol was more effective in increasing the quadriceps or hamstring muscle strengths. A larger sample group and a more objective measure to quantify the presence and extent of arthrogenic muscle inhibition and its' effect on muscle strength should be considered for future studies investigating the effect of manipulative therapy on muscle strength.

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CHAPTER ONE

INTRODUCTION

1.1 THE PROBLEM

Knee joint pathologies, in general, are associated with a loss of knee-extensor muscle strength. This weakness has been attributed to arthrogenic muscle inhibition (AMI) (Suter *et al.*, 2000).

AMI is defined as the inability of a muscle to recruit all motor units of a muscle group to their full extent during a maximal effort voluntary muscle contraction and is a natural response designed to protect the joint from further damage (Suter *et al.*, 2000). Mechanoreceptor activity plays the primary role in AMI (Hopkins and Ingersoll, 2000).

AMI therefore results from the activity of many different mechanoreceptors within a joint, namely: Ruffini endings, Golgi-like endings and Pacinian corpuscles. Pain receptors such as free-nerve endings are found throughout the joint tissue and are sure to be active with any joint damage. It is unknown as to whether they play a significant role in AMI. These mechanoreceptors act on inhibitory interneurons synapsing on the motor neuron (MN) pool of the joint musculature, decreasing the force of any contraction stemming from that MN pool. (Hopkins and Ingersoll, 2000). These mechanoreceptors are located in joint capsules, ligaments and tendons (Levangie and Norkin, 2001:71).

The concept that spinal manipulation could somehow influence, or even improve on, the function of the nervous system, still remains within the realms of philosophy (Wyke, 1985: 72-77), but the neurophysiological effects of spinal manipulation have been observed in body segments distant from where the manipulations were performed (Suter *et al.*, 1994; Suter *et al.*, 1999; Suter *et*

al., 2000; Naidoo, 2002). Wyke (1985: 72-77) noted that articular mechanoreceptor afferent nerve fibers give off collateral branches that are distributed both intersegmentally and intrasegmentally. Therefore manipulation of an individual joint not only affects the motor unit activity in the muscles operating over the joint being manipulated, but also in more remote muscles.

The sacroiliac (SI) joint has been linked to MI within the quadriceps muscle group (Suter *et al.*, 1999; Suter *et al.*, 2000; Sakamoto *et al.*, 2001).

Structures within the knee joint that have been shown to induce quadriceps MI are the lateral and medial collateral ligaments (Kim *et al.*, 1995), the anterior and posterior cruciate ligaments (Fisher-Rasmussen *et al.*, 2002; Lewek *et al.*, 2002), the joint capsule (Hopkins *et al.*, 2001), perimeniscal tissue and the outer third of the menisci (Hopkins and Ingersoll, 2000). The lateral retinaculum has also been identified as a possible source of AMI although it is not known whether it plays a significant role (Hopkins and Ingersoll, 2000; Sanhis-Alfonso and Rosello-Sastre, 2000). Immobilization and effusions of the knee are also associated with quadriceps MI (Cruz-Martinez, Ramirez and Arpa, 2000; Hopkins and Ingersoll, 2000; Hopkins *et al.*, 2001).

Two studies showed that manipulation of the SI joint resulted in an increase in quadriceps muscle strength (Suter *et al.*, 1999, Suter *et al.*, 2000). The effect of manipulating the knee joint on quadriceps muscle strength however has not yet been investigated, to the best knowledge of the researcher.

1.2 THE STATEMENT OF THE PROBLEM

This study proposes to investigate the effect of three manipulative treatment protocols on quadriceps muscle strength in patients with Patellofemoral Pain Syndrome.

1.2.1 Objective One

The first objective was to evaluate the effect of the long axis distraction manipulation of the tibio-femoral joint on quadriceps muscle strength, utilizing the Cybex Orthotron II Isokinetic Rehabilitation System.

1.2.2 Objective Two

The second objective was to determine the effect of manipulating the sacroiliac joint on quadriceps muscle strength, utilizing the Cybex Orthotron II Isokinetic Rehabilitation System.

1.2.3 Objective Three

The third objective was to determine the effect of manipulating both the tibio-femoral and sacroiliac joints on quadriceps muscle strength, utilizing the Cybex Orthotron II Isokinetic Rehabilitation System.

1.2.4 Objective Four

The fourth objective was to evaluate the effect of the three manipulative treatment protocols on the subjective perception of anterior knee pain, utilizing the Numerical Pain Rating Scale.

Subsequent to beginning the study, research was published identifying the presence of hamstring weakness in patellofemoral pain syndrome patients (Clifton, 2003). While this study was not originally concerned with the hamstring muscle, the concentric-concentric isokinetic testing of the thigh muscle made gathering of data possible. Any possible effect of manipulating the tibio-femoral or sacroiliac joint on the hamstring was measured and quantified in a similar manner to that for the quadriceps.

1.3 ASSUMPTIONS

The following assumptions were made in the study:

1. Suter *et al.* (2000) stated that arthrogenic muscle inhibition is the inability of a muscle to recruit all motor units of a muscle group during a maximal effort voluntary muscle contraction. Therefore, recruitment of an inhibited muscle group does not occur to its full extent.
2. Hopkins and Ingersoll (2000) believe that mechanoreceptor activity plays the primary role in arthrogenic muscle inhibition. Manipulation¹ of a joint has been proposed to activate mechanoreceptors from structures in and around the manipulated joint. The altered afferent input arising from the stimulation of these receptors is thought to cause changes in the motor neuron excitability, with a subsequent decrease in AMI (William, 1997: 144; Suter *et al.*, 2000).

¹ A manipulation is defined as a small amplitude, high velocity thrust at the end of the elastic barrier of the joint capsule (William, 1997: 144).

1.4 POTENTIAL BENEFITS OF THE STUDY

This research will add to the body of knowledge regarding the effects of manipulation on quadriceps muscle strength in patients suffering from Patellofemoral Pain Syndrome. This knowledge could be used to provide effective treatment and rehabilitation protocols for knee injuries associated with Patellofemoral Pain Syndrome (Hopkins and Ingersoll, 2000).

Muscle inhibition and a decrease in muscle strength hinders early active exercise in the joint rehabilitation process, which is essential for decreased healing time, increased vascular in growth, quicker regeneration of scar tissue, and stronger ligament and tendon healing. A therapeutic intervention that could block or slow arthrogenic muscle inhibition would allow clinicians to return an athlete to participation with less strength or kinesthetic limitations once healing has occurred (Hopkins and Ingersoll, 2000).

After rehabilitation for knee injuries has been completed, athletes often return to competition deficient in strength and neuromuscular control, and this results in an increased susceptibility to re-injury of the rehabilitated area (Hopkins and Ingersoll, 2000). Suter *et al.*, (2000) showed that there was an increase in quadriceps muscle strength following sacro-iliac joint manipulation. If manipulation of the knee joint has a synergistic effect on quadriceps muscle strength, then the amount of strength regained by the athlete (before returning to competition) could be increased, potentially reducing the chance of re-injury (Hopkins and Ingersoll, 2000).

CHAPTER TWO

THE LITERATURE REVIEW

2.1 INTRODUCTION

This chapter reviews the relevant literature pertaining to this study. Information was obtained from journals, articles, published reports, web sites and textbooks.

The following aspects were reviewed:

- Anatomy and functional biomechanics of the knee and sacroiliac joints,
- Anatomy of the thigh,
- All relevant neuroanatomy,
- Patellofemoral pain syndrome,
- Joint dysfunction and its relationship to arthrogenic muscle inhibition.

2.2 ANATOMY AND FUNCTIONAL BIOMECHANICS

2.2.1 The knee

2.2.1.1 Introduction

The knee is a highly modified hinge joint that joins the femur to the tibia and the patella. In spite of the presence of menisci, the knee is an incongruent joint, which has successfully amalgamated the properties of mobility and stability (Segal and Jacob, 1984:9). The knee is made up of three joints: the tibiofemoral, patellofemoral and superior tibiofemoral joints (Moore, 1982:477-487). The tibiofemoral joint is composed of the lower end of the femur, the upper end of the tibia, the upper end of the fibula and the patella. The femoral condyles form a hollow area in which the patella lies, forming the patellofemoral joint. This planer joint permits the patella to glide over the surface of the femur during flexion and extension (Austermuehle, 2001). The

superior tibiofibular joint is a synovial joint between the head of the fibula and the lateral condyle of the tibia (Moore, 1992: 486).

2.2.1.2 Ligaments of the knee

Local thickenings around the fibrous capsule of the knee form the extrinsic ligaments of the knee. The patella, medial and lateral collateral and the oblique and arcuate popliteal ligaments make up the extrinsic ligaments of the knee. The anterior and posterior cruciate ligaments are the intra-synovial ligaments of the knee, as they are internal to the fibrous capsule (Moore, 1992: 477-478).

2.2.1.3 Innervation

The segmental supply of the knee is derived from L2-4 in the form of articular nerve branches from the obturator, femoral, tibial and common fibular nerves (Solomon, Schmidt and Adragna, 1990: 491; Moore, 1992: 486).

2.2.1.4 The Patella

The patella is the largest sesamoid bone in the body and is located on the anterior aspect of the knee within the tendon of the quadriceps femoris muscle. It has a triangular shape. The base of the patella is broad and is directed superiorly while the apex is pointed and is directed inferiorly. The posterior surface of the patella is divided into two articular surfaces by a bony ridge (Solomon, Schmidt and Adragna, 1990: 248-249). Seven facets are located on the posterior surface of the patella and they form the articulation between the patella and the sulcus of the femur. There are three facets on the medial and lateral surfaces of the patella respectively and an extra "odd" facet on the medial side. The surface anatomy of each side, the overall functional anatomy of the entire lower extremity and the relationship of the surrounding muscles affect the contact area between the patella and femur (Tria, Palumbo and Alicea, 1992).

The patella increases the lever arm for the quadriceps / patella tendon by up to 50%, effecting knee extension and resisting knee flexion. It centralizes the efforts of divergent muscle groups of the quadriceps and protects the knee joint anteriorly. Passive stabilizers of the patella include the trochlea of the femur, the peripatella retinaculum and the shape of the patella. The patella is dynamically stabilized by the pes anserinus and semi-membranous muscles, rotating of the tibia inward; the biceps femoris muscle, rotating the tibia outward; the vastus medialis muscle, pulling the patella medially; the vastus lateralis muscle, pulling laterally; and the vastus intermedius and rectus femoris muscles, pulling proximally and laterally (Thomee, Augustsson and Karlsson, 1999).

The lower fibres of the vastus medialis muscle, the vastus medialis oblique, and the anterior projection of the lateral femoral condyle, appear to play the most important role in resisting the lateral pull created by the Q-angle during extension of the knee (Reid, 1992: 438; Norris, 1998: 240; Sanchis-Alfonso, Rosello-Sastre and Martinez-Sanjuan, 1999)

2.2.1.5 Biomechanics

2.2.1.5.1 Stability

The capsule, ligaments and muscular system give the joint its stability, which is most obvious during complete extension of the knee. Mobility of the knee joint is greatest during flexion (Reid, 1992: 437-438). The tibiofemoral joint has physiological movements of flexion and extension and a small range of rotation. Accessory movements include abduction, adduction, rotation, and posteroanterior, anteroposterior, medial, lateral and cephalid and caudal longitudinal movements (Maitland, 1991: 238).

2.2.1.5.2 The Q-angle

The Q-angle is an overall measurement of patella alignment. It is the angle between a line from the anterior superior iliac spine to the centre of the patella and a line from the centre of the patella through the centre of the tibial tubercle. Normal values for the Q-angle are in the region of 15 – 20 degrees (Reider, 1999: 213). As the knee flexes and extends, the patella should travel in line with the long axis of the femur. However, the horizontal vector created as a result of the Q-angle tends to pull the patella laterally. This movement is resisted by the vastus medialis oblique (Norris, 1998: 240). The Q-angle will increase with femoral neck anteversion, external tibial torsion, a wider female pelvis, genu valgum (knock-knees), tightness of the tensor fascia lata and the iliotibial band or abnormal attachment of the iliotibial band, weakness of the gluteus medius and foot pronation. An increase in the Q-angle may be associated with the tendency towards developing patellofemoral pain (Sanchis-Alfonso, Rosello-Sastre and Martinez-Sanjuan, 1999).

2.2.1.5.3 Patellofemoral Joint Reaction Forces

The patellofemoral joint reaction force (PJRF) is the vector resultant from the quadriceps tension and patella tendon tension acting perpendicular to the articular surfaces. The PJRF is a measure of the compression of the patella against the femur and is dependant on the angle of knee flexion, as well as on muscle tension (Sanchis-Alfonso, Rosello-Sastre and Martinez-Sanjuan, 1999).

In full extension, the patella does not contact the femur and therefore, the PJRF increases with increasing flexion. This occurs because the angle between the tendon and the quadriceps becomes more acute, and as knee flexion increases, the effect of the lever arms of the femur and tibia increase (Sanchis-Alfonso, Rosello-Sastre and Martinez-Sanjuan, 1999). Patellofemoral compression forces increase with increasing knee angles-up to 90⁰ of knee flexion-and can reach up to 8 times bodyweight (Thomee, Augustsson and Karlsson, 1999). Patellofemoral loads may be as high as 3 to 4 times bodyweight as the

knee flexes in walking, and 9 times bodyweight when descending stairs. Classical pain-provoking activities of daily living are; ascending or descending stairs, squatting, riding a bicycle, and sitting for prolonged periods of time with bent knees (Norris, 1998: 240-241; Sanchis-Alfonso, Rosello-Sastre and Martinez-Sanjuan, 1999).

Intermittent compression of the patella is required for adequate nutrition of hyaline cartilage, but the question remains as to when this compression becomes excessive (Reid, 1992: 347).

2.2.1.5.4 Patella tracking

The normal movement of the patella during knee flexion and extension follows an S-shaped curve through the trochlea. During extension of the knee, from approximately 30° of knee flexion, the tibia rotates outwards and the patella is guided through the trochlea of the femur by the interacting heads of the quadriceps muscle group. At full extension, the patella rests on the supra-patella fat pad. During knee flexion from the extended position, the distal part of the patella comes in contact with the lateral femoral condyle at 10 to 20° of knee flexion. As the knee continues on to 90° of flexion, the area of contact on the patella moves upward, failing to reach the extreme medial or “odd” facet. At the same time, the patella demonstrates lateral movement, tending to leave some of the medial femoral condyle uncovered. As the movement continues to 135°, the lateral shift continues so that at its completion the medial patella facet lies free in the intercondylar notch and the “odd” facet contacts the lateral aspect of the medial femoral condyle. (Reid, 1992: 347-348; Thomee, Augustsson and Karlsson, 1999).

Altered patella tracking occurs when the patella is pulled laterally as the knee is straightened under load. Proper tracking of the patella during flexion and extension is influenced by a number of factors. (Davidson, 1993; Reive, 2000):

- The height of the femoral condyles and hence the depth of the sulcus which keeps the patella seated and tracking properly.
- The shape of the facets on the undersurface of the patella, which helps to determine the “fit” between the patella and the femoral groove.
- The medial and lateral retinacula, which keep the patella centred between the femoral condyles during movement.
- The composite angle of the pull of the quadriceps group (the Q-angle).
- Relative strength of individual muscles of the quadriceps group.
- Sufficient strength of the abductor muscles is essential to prevent excessive rotation in the femur.
- Any abnormality of the anatomical structures influencing the patella movement can cause excessive pressure between the patella and the femoral condyles.

2.2.2 The sacroiliac joint

2.2.2.1 Introduction

The sacroiliac (SI) joint is a strong synovial joint between the articular surfaces of the sacrum and ilium. These surfaces have irregular elevations and depressions, which result in partial interlocking of these bones (Moore, 1992: 251). The SI joint is usually auricular or C-shaped, with the convex contour facing anteriorly and slightly inferiorly (Cassidy and Mierau, 1992: 211).

There can be marked variations in the size, shape and contour of the SI joint. The vertically oriented auricular surface lies obliquely at an angle to the sagittal plane (Cassidy and Mierau, 1992: 211). The joints of the male have extra- and intra-articular tubercles and are built for strength, whereas the female articulation is built for mobility and parturition (Walters, 1993: 150).

The SI joint is a synovial joint, where the articular surfaces of the bones are covered with hyaline cartilage and are united by an articular capsule. The hyaline cartilage on the sacral surface is three times as thick as the fibro cartilage on the iliac surface. A synovial membrane lines the inner surface of the capsule and produces synovial fluid for lubrication of the joint cavity. (Walters, 1993: 150-151).

2.2.2.2 Ligaments

The SI joint is a weight-bearing joint that is stabilized by a series of very strong ligaments (Cassidy and Mierau, 1992: 211). The interosseous SI ligament unites the iliac and sacral tuberosities and is supported by the posterior SI ligament. The posterior ligament is composed of:

1. Strong, short transverse fibres that join the ilium and first and second tubercles of the lateral crest of the sacrum,
2. Long vertical fibres that the third and fourth transverse tubercles of the sacrum to the posterior iliac spines.

The posterior ligament blends in with the sacrotuberous ligament (Moore, 1992: 251).

The anterior SI ligament is a thickening of the anterior and inferior parts of the fibrous capsule. It is thickest where it connects the sacrum and ilium at the third sacral segment. The iliolumbar, sacrotuberous and sacrospinous ligaments all support the SI joint and are termed accessory ligaments. The interosseous, posterior and anterior SI ligaments are known as capsular ligaments. (Walters, 1993: 151).

2.2.2.3 Innervation

Segmental supply of the SI joint can range from L2-S4. The L4 and L5 levels most frequently innervate the anterior aspect of the joint, whilst the posterior aspect more commonly receives innervation from S1 and S2 levels (Cassidy

and Mierau, 1992: 211-212). Plexiform networks from the posterior primary rami of the L5-S4 segments innervate the posterior aspect of the SI joint. The accessory ligaments of the SI joint receive fibres from L1-S2. (Walters, 1993: 152-153).

Mechanosensitive afferent units have been identified in the SI joint and adjacent tissues (Sakamoto *et al.*, 2001). Most of these units are nociceptive receptors (Cassidy and Mierau, 1992: 211-212; Sakamoto *et al.*, 2001).

2.2.2.4 Biomechanics

Although many attempts have been made to describe and measure the motion in the SI joint, its biomechanical function remains largely unknown (Cassidy and Mierau, 1992: 215). The SI joint possesses little mobility. The articular surfaces of the sacrum and ilium are irregular and fit together securely. This arrangement prevents easy dislocation and lessens the strain on the supporting ligaments of the joint. Transmission of body weight to the hips is facilitated in this manner (Moore, 1992: 251). Motion in this joint is limited and is dependent on some degree of joint separation. Joint movements are coupled, consisting mainly of x-axis rotation and z-axis translation (Cassidy and Mierau, 1992: 215).

The SI joint is surrounded by some of the largest and most powerful muscles in the body, but none of these cross the joint or are known to directly influence joint movement. However, contraction of these muscles (erector spinae, psoas, quadratus lumborum, piriformis, rectus femoris and gluteus maximus, minimus and medius) will place shear and moment loads on the joint surfaces, hence influencing any movement at the SI joint. (Cassidy and Mierau, 1992: 215). The quadriceps and hamstring muscles also play an important role in ensuring normal SI movements (Walters, 1993:155).

2.2.3 The thigh

The following muscles span the SI and knee joints and are related, directly or indirectly, to movements of the knee and hip/sacroiliac joint.

The quadriceps femoris muscle is made up of four heads, namely the rectus femoris, vastus lateralis, vastus medialis and vastus intermedialis. The semitendinosus, semimembranosus and biceps femoris are collectively known as the hamstring muscles (Moore, 1992: 368-387; 241-243).

The following table summarizes the origin, insertion and innervation of the muscles listed above.

Table 2.1: Summary of the quadriceps and hamstring muscles.

| Muscle | Origin | Insertion | Innervation |
|------------------------------|---|--|---|
| THE QUADRICEPS MUSCLE | | | |
| Rectus Femoris | Anterior iliac spine and groove superior to the acetabulum. | Base of the patella and via the patella ligament to the tibial tuberosity. | Femoral nerve L2-L4. |
| Vastus Lateralis | Greater trochanter and lateral lip of the linea aspera of the femur. | | |
| Vastus Medialis | Intertrochanteric line and medial lip of the linea aspera of the femur. | | |
| Vastus Intermedialis | Anterior and lateral surface of body of the femur. | | |
| THE HAMSTRINGS | | | |
| Semitendinosus | Ischial tuberosity. | Medial surface of the superior part of the tibia. | Tibial division of the sciatic nerve L5-S2. |
| Semimembranosus | | Posterior part of the medial condyle of the tibia. | |
| Biceps femoris | <u>Long head</u> - Ischial tuberosity. <u>Short head</u> - Lateral lip of the linea aspera and lateral supracondylar line. | Lateral side of the fibula. | <u>Long head</u> - tibial division of sciatic nerve L5-S2. <u>Short head</u> - peroneal division of the sciatic nerve L5-S2. |

(Moore, 1992: 387 and 423)

2.3 RELEVANT NEUROANATOMY

2.3.1 Introduction

The spinal cord consists of a complex system of channels that relay information from several parts of the body. The central and peripheral nervous systems work together to gather, transmitting and processing information from many different neurophysiological systems in order to coordinate movement (Hopkins and Ingersoll, 2000). A review of the neuroanatomy involved in the transmission of this information follows.

2.3.2 Joint receptors

2.3.2.1 Intra-articular receptors

Receptors are specialized cells that change their properties in response to specific stimuli of various types. Receptors that respond to physical or mechanical stimuli are termed mechanoreceptors. Mechanoreceptors act to transduce energy from one form, for example tension, into a specific nerve signal. Receptors that transduce information about the relationship between body segments are proprioceptors. Joint receptors are mechanoreceptors and can also act as proprioceptors. Therefore, joint receptors have two major functions. They provide position sense or information about the relative configuration of body segments, and they initiate protective reflex mechanisms that protect and help stabilize the joint. (Hopkins and Ingersoll, 2000).

The four different types of mechanoreceptors in the knee joint are:

Type 1- Ruffini bodies.

Type 2- Vater-Pacini bodies.

Type 3- Golgi organellae.

Type 4- Free nerve endings.

(Freiwald, Reuter and Engelhardt, 1999: 83)

2.3.2.1.1 Type 1- Ruffini bodies

These are, after free nerve endings, the second most frequent receptor type in the knee joint. They are arranged in clusters of 3 to 6 bodies. (Freiwald, Reuter and Engelhardt, 1999: 83). Ruffini bodies are slow-adapting receptors that have been identified in the joint capsule, anterior cruciate ligament and in the perimeniscal tissue and outer third of the meniscus. These receptors have a very low threshold and they respond to very slight changes in ligament tension and capsular pressure. (Hopkins and Ingersoll, 2000).

2.3.2.1.2 Type 2- Vater-Pacini bodies

This receptor type has been found in the fibrous knee joint capsule, Hoffa's infrapatella fat body and the vascularized area of the medial meniscus, and ligament insertions. The function of the Vater-Pacini bodies is the rapid uptake and transport of information from the afferent-supply tissues. They are inactive in immobile joints and likewise when the knee joint is moved at a constant velocity. They become active in the case of accelerations or decelerations. Vater-Pacini sensors have a low mechanical stimulation threshold, adapt rapidly and they act as dynamic mechanoreceptors. (Freiwald, Reuter and Engelhardt, 1999: 83).

2.3.2.1.3 Type 3- Golgi organellae

The Golgi tendon organs in the muscle-tendon bond are identical to those in other tissues of the knee joint. Golgi organs are similar to the Ruffini bodies and as long as they are localized in the joints, some authors do not differentiate between them. Golgi organs are found in the medial and lateral collateral ligaments, the anterior and posterior cruciate ligament, the medial and lateral meniscus, and in the muscle-tendon configuration of the patella tendon. These receptors help provide information about joint position. (Freiwald, Reuter and Engelhardt, 1999: 83-84).

2.3.2.1.4 Type 4- Free nerve endings

Free nerve endings are non-specialized, non-encapsulated, unmyelinated (or finely myelinated) receptors. They function as pain receptors and probably provide a crude awareness of initial joint movement. These nerve endings are found throughout the joint tissue and are involved with any joint damage. (Hopkins and Ingersoll, 2000).

2.3.2.2 Extra-articular receptors

Muscle spindles are extra-articular receptors that are located within skeletal muscle and run parallel to the skeletal muscle fibres. The fibres in the centre of the muscle spindle are known as intrafusal fibres, whilst the skeletal muscle fibres surrounding the muscle spindle are known as the extrafusal fibres. The intrafusal fibres are supplied by type I alpha nerve fibres, which stimulate motor neurons. (Solomon, Schmidt and Adragna, 1990: 553-554). The afferents of the muscle spindle are type II nerve fibres that synapse with spinal ganglia cells. Type II afferents also stimulate the motor neurons but their effect is multi-segmental. They have a promoting effect on the agonist and an inhibitory effect on the antagonist muscle. (Freiwald, Reuter and Engelhardt, 1999: 85-86).

Stimulation of the muscle spindle due to stretching of that muscle causes a reflex contraction. This reflex is known as the stretch reflex and brings the tension in the muscle back to normal (Solomon, Schmidt and Adragna, 1990: 554). Mechanoreceptors and central nervous system mediation can also stimulate the muscle spindle fibres. Hence, muscle spindles either directly or indirectly influence the motor neurons of the agonistic and antagonistic musculature. (Freiwald, Reuter and Engelhardt, 1999: 86).

2.3.3 Neural pathways

2.3.3.1 The afferent pathway to the spinal cord

Most receptors are specialized endings to sensory nerve fibres (Hopkins and Ingersoll, 2000). Sensory nerve cells contain a cell body located in a dorsal root ganglion-very close to the spinal cord. The cell body then projects through the dorsal horn of the spinal cord where it can make connections with several different types of neurons. (Haldeman, 1992: 171-172).

Sensory distribution of the knee is divided into two groups: an anterior group and a posterior group (Hopkins and Ingersoll, 2000), with

- (1) The anterior group of afferents supplying the joint capsule, collateral ligaments and patella tendon.
- (2) The posterior group of afferents supplying the posterior capsule, the outer portion of the menisci, the cruciate ligaments and the infrapatella fat pad.

2.3.3.2 The interneuron

Once the sensory fibre enters the dorsal horn of the spinal cord, it usually branches to synapse on several interneurons. An interneuron is defined as a neuron receiving information from a neuron and transmitting it to other neurons. A single neuron can receive information from many other neurons and project it to many different neurons. Most interneurons have axons that branch widely, ascending or descending in the white matter over distances of 2-3 segments before re-entering the grey matter. (Hopkins and Ingersoll, 2000).

Interneurons are the intermediates of pathways to alpha and gamma motor neurons (MN), to autonomic efferent neurons and to ascending pathways. They receive projections from sensory afferent fibres, descending fibres and other interneurons (Hopkins and Ingersoll, 2000). Processing of afferent input

into the spinal cord is mediated by inputs from other parts of the periphery and from the brain (Haldeman, 1992: 173). Corticospinal, rubrospinal and vestibulospinal tracts give input to the interneuron. Peripheral afferent information that gives input to the interneuron comes from muscle spindles, Golgi tendon organs, joint, and cutaneous receptors. The net effect of all information arriving at the interneuron is expressed in the inhibitory or excitatory response of the MN pool. (Hopkins and Ingersoll, 2000).

2.3.3.3 The ascending pathway

Sensory information enters the spinal cord through the dorsal roots via the afferent fibres branch and ascends via the dorsal column pathway. The dorsal column pathway also receives fibres from other interneurons. The ascending fibres terminate in the medulla, continuing via the medial lemniscus, to the ventropostero-lateral nucleus of the thalamus and on to the cerebral cortex. Descending pathways are arranged into spinal tracts that carry specific information from a supraspinal centre. (Hopkins and Ingersoll, 2000).

2.3.3.4 The descending pathway

2.3.3.4.1 The corticospinal tract

Cortical neurons synapse on alpha motor neurons (MNs), gamma MNs, and interneurons. Cortical neurons carry motor information to the MNs. Most MNs facilitate, although some cortical neurons are inhibitory. Lesions to peripheral mechanoreceptors functionally modify the somatosensory pathways, which carry this information. Although it has been suggested that the cortex is involved in complex spatial integration of articular proprioceptive inputs, it has been shown that joint afferents do not change cortex activity. The role of the corticospinal system in arthrogenic muscle inhibition (AMI) is not completely understood. (Hopkins and Ingersoll, 2000).

2.3.3.4.2 The vestibulospinal tract

The vestibulospinal tract regulates postural reflexes through projections to MNs and interneurons. The vestibulospinal tract remains tonically active to help maintain upright posture. (Hopkins and Ingersoll, 2000).

2.3.3.4.3 The rubrospinal tract

Like the corticospinal tract neurons, neurons from the rubrospinal tract innervate MNs and interneurons controlling distal musculature. The rubrospinal tract has also been implicated in inhibitory actions affecting interneurons. (Hopkins and Ingersoll, 2000).

Neurons from supraspinal centres converge on inhibitory interneurons to inhibit the inhibitory mechanism. During joint injury, descending spinal inhibition is reduced, allowing for an increase in AMI. (Hopkins and Ingersoll, 2000).

2.4 PATELLOFEMORAL PAIN SYNDROME

2.4.1 Definition

Patellofemoral pain syndrome (PFPS) is defined as the presence of pain around the patella, which is associated with activities that load the patellofemoral joint. PFPS is an umbrella term used to encompass all anterior or retropatella pain in the absence of other specific pathology. (Crossley *et al.*, 2001).

Other names for PFPS include: retropatella pain syndrome, patellofemoral arthralgia, extensor mechanism disorder, lateral patella compression syndrome, patellalgia, patellofemoral dysfunction and anterior knee pain (Davidson, 1993; Thomee, Augustsson and Karlsson, 1999).

The term anterior knee pain is suggested to encompass all pain related to problems of the anterior part of the knee. By excluding anterior knee pain due to intra-articular pathology, peripatella tendonitis or bursitis, plica syndromes, Sinding-Larsen's Johanssen's disease, Osgood Schlatter's disease, neuromas, and other rarely occurring pathologies, it is suggested that the remaining patients with anterior knee pain be diagnosed with PFPS. PFPS seems to be an appropriate term as no distinction can be made as to which specific structure of the patella or the femur is affected. "Pain" is a symptom that all patients experience, but patients also have other symptoms, thus it is appropriate to use the word "syndrome". (Thomee, Augustsson and Karlsson, 1999).

PFPS is now used in preference to the term "Chondromalacia Patella", which in the past has been used as a synonym for PFPS. According to the International Patellofemoral Study Group, the term chondromalacia should not be used to describe a clinical syndrome, but should be used as a descriptive term for morphological softening of the patella cartilage. (Sanchis-Alfonso, Rosello-Sastre and Martinez-Sanjuan, 1999).

2.4.2 Incidence

The most common overuse running injury 20 years ago was PFPS; this is still the case today (Tauton *et al.*, 2002). PFPS remains one of the most common musculoskeletal entities encountered by physiotherapists and sports medicine practitioners (Crossley *et al.*, 2002).

PFPS is the most common knee complaint seen in adolescents and young children (Witvrouw *et al.*, 2000). Among injuries to physically active adolescents and young adults, knee problems are one of the most common, of which PFPS is the most frequent complaint. Tauton *et al.* (2002), in a retrospective analysis of running injuries, found that PFPS was the most common overuse syndrome and that this type of injury was significantly more common in females. Salem and Powers (2001) support this finding in their review of the National Collegiate Athletic Association Injury Surveillance System (USA).

In clinics dealing with musculoskeletal complaints, PFPS may account for almost 10% of visits and 20-40% of all knee complaints (Kannus *et al.*, 1999: 356). Arroll *et al.* (1997) cited two studies where 35% of all patients seen at a Finish sports clinic and 15% of Israeli military recruits suffered from PFPS. In one British sports injury clinic, 5.4% of total injuries seen and 25% of all knee problems treated over a five-year period were attributed to PFPS (Davidson, 1993).

In reports from different sports medicine clinics, knee complaints account for 23-31% of injuries, where pain conditions related to the patellofemoral joint are the most common. (Thomee, Augustsson and Karlsson, 1999). People under the age of 34 years (Taunton *et al.*, 2002), and athletes who participate in sports involving jumping and running activities are at the greatest risk for developing patellofemoral-related injuries (Salem and Powers, 2001).

2.4.3 Natural history

Spontaneous resolution of pain may occur (Juhn, 1999). Many individuals with PFPS will experience a return of their symptoms at some point in time (Thein, Brody and Thein, 1998). PFPS frequently becomes chronic and the pain may force the patient to limit physical activity (Kannus *et al.*, 1999). There is little research that shows a progression of PFPS to patellofemoral osteoarthritis (Thomee, Augustsson and Karlsson, 1998; Kannus *et al.*, 1999). PFPS is self-limiting and a reduction in activity levels corresponds to a reduction in most symptoms associated with PFPS (Reid, 1992: 364).

In a study by Sandow and Goodfellow (1985) on 62 girls aged between 14 and 16 years, a follow-up between 2 to 8 years after initial consultation was performed. The following interventions were used: physiotherapy, plaster immobilization for short periods of time, arthroscopy and double-contrast arthrograms. The treatment protocols were not uniform, throughout the sample group, in terms of interventions applied and number of treatment visits. Up to 30% of patients were only seen once. Questionnaires were submitted to all participants, of which 54 were returned. The results based on an 86% response where as follows:

- (1) 94.4% of respondents continued to experience some pain, between 46.3% reported diminished severity, while only 13% reported worse symptoms.
- (2) 81.5% reported pain frequency of once a week or less and 87% of patients rarely or never used analgesics.
- (3) 48.1% reported knee pain that did not interfere with sport at all.

(Sandow and Goodfellow, 1985)

The study by Sandow and Goodfellow (1985) suggests that PFPS is therefore a benign condition, which affects the individual for many years after the initial onset. Pain was less intense and less frequent, and limited participation in

sport seemed to result. Similar results were discussed by Clifton's (2003) review of Blond and Hanse's 5.7-year review of PFPS in 250 athletes.

Kannus *et al.* (1999), in a 7-year follow up study, used bone densitometry, radiography and magnetic resonance imaging (MRI) and found that PFPS did not lead to patellofemoral osteoarthritis or osteopenia. Therefore Kannus *et al.* (1999) stated that only a 10-20 year follow up study would provide a clear picture of the natural history of PFPS.

2.4.4 Pathophysiology

In the review of the current literature there is little consensus as to the origin of pain in PFPS patients. This problem is reflected by the many terms used to describe anterior knee pain. (Reid, 1992: 350).

Currently, there are two theories that explain the origin of anterior knee pain:

- (1) The neural theory and
- (2) The mechanical theory.

Both theories are not exclusive but complementary (Sanchis-Alfonso, Rosello-Sastre and Martinez-Sanjuan, 1999).

2.4.4.1 The neural theory

The neural theory investigates the role of neural damage in the symptoms of PFPS. This theory has received little attention in the literature so far.

With chronic lateral subluxation of the patella, as seen in PFPS patients, shortening of the lateral retinaculum occurs. With knee flexion, the patella migrates medially into the femoral condyle, which places the lateral retinaculum under excessive stress. This produces a recurrent stretch on the lateral retinaculum that can cause secondary nerve changes. The neural changes observed resemble those of neuromas (Davidson, 1993; Sanchis-

Alfonso, Rosello-Sastre and Martinez-Sanjuan, 1999). Sanchis-Alfonso, Rosello-Sastre and Martinez-Sanjuan (1999) found no other histological or inflammatory changes justify the pain in PFPS patients.

Sanchis-Alfonso, Rosello-Sastre and Martinez-Sanjuan (1999) found that substance-P- a nociceptive neurotransmitter- was present in some patients with moderate levels of PFPS and absent in severe PFPS patients. Substance-P fibres have been identified in the lateral retinaculum, fat pad, synovial membrane, menisci and the subchondral plate of patellae affected by degenerative disease (Sanchis-Alfonso and Rosello-Sastre, 2000). Sanchis-Alfonso, Rosello-Sastre and Martinez-Sanjuan (1999) postulate that pain begins with a proliferation of tiny perivascular nerves capable of releasing substance-P. If the lesion advances, the damaged nerves undergo a neuromatous transformation with fibrosis and resultant loss of the small nerve fibres and loss of the neurotransmitory ability. Hence if, the knee injury persists, the presence of substance-P will decrease and eventually disappear.

Along with the neural changes discussed, proprioceptive feedback from the knee may also be affected in PFPS patients. In PFPS patients, nerve damage occurs diffusely in the affected retinaculum (Sanchis-Alfonso, Rosello-Sastre and Martinez-Sanjuan, 1999), therefore one must consider the possibility of multiple neurological sequelae in the peripatella region, including altered proprioceptive innervation. Proprioceptive feedback from joint mechanoreceptors plays an important role in proper knee function. (Norris, 1998:34). Instability sometimes associated with PFPS might be explained, at least in part, as a result of altered or loss of joint afferent proprioceptive information (Sanchis-Alfonso, Rosello-Sastre and Martinez-Sanjuan, 1999).

2.4.4.2 The mechanical theory

The mechanical theory stresses repetitive micro trauma (overuse) and abnormal patella tracking as major contributing factors to the development of pain in the PFPS patients (Davidson, 1993). Not only does the patella move up and down, it also tilts and rotates, so there are various points of contact

between the undersurface of the patella and the femur. Repetitive contact at any of these areas, sometimes combined with maltracking of the patella, is the likely mechanism of patellofemoral pain. PFPS is often classified as an overuse syndrome, but should perhaps be more appropriately termed an overload syndrome because PFPS may also affect inactive patients. (Juhn, 1999).

Pain in PFPS patients may be due to excessive compression forces between the patella and the femoral condyles during knee flexion and extension (Austermuhle, 2001). This compressive force is also known as the patellofemoral joint reaction (PFJR) force. These forces may be produced either by activities that augment flexion of the knee or by direct trauma. These activities are common in both sport and daily living. (Sanchis-Alfonso, Rosello-Sastre and Martinez-Sanjuan, 1999).

When the PFJR force is not adequately dispersed across the entire surface area of the patella cartilage, damage to this articular cartilage may occur. Two distinct lesions may affect the articular cartilage:

- The first type is surface degeneration of the hyaline cartilage (Reid, 1992: 350). The hyaline cartilage is aneural, therefore changes in the patella cartilage itself would not result in anterior knee pain (Norris, 1998: 242).
- The second lesion appears to be initiated in the deep layers of the cartilage and is characterized by basal degeneration. Tangentially-orientated collagen bundles at the articular surface and more vertically-orientated fibres down to the subchondral bone characterize normal articular cartilage. When the articular cartilage is subjected to excessive compression, there is disorganization of the intermediate and deep layers and subsequent softening of the cartilage. The cushioning effect of the cartilage is diminished, resulting in stimulation of nociceptive fibres in the subchondral bone. In PFPS the presence of a ridge across the medial femoral condyle causes friction with the odd

facet, as the patella rides the ridge during knee movements. The odd facet of the patella bears weight infrequently during the day, but when it is loaded, it is usually under circumstances that create high forces (ie: any activity that involves prolonged or extreme flexion of the knee). This stress will cause disorganization and softening of the intermediate and deep cartilage layers. This change may lead to a situation where the articular surface appears grossly smooth but is not doing its job as a stress-reliever for the underlying bone. (Reid, 1992: 351-354; Sanchis-Alfonso, Rosello-Sastre and Martinez-Sanjuan, 1999).

The byproducts of cartilage degeneration may produce a chemical synovitis and this could explain the popliteal pain in some PFPS patients (Sanchis-Alfonso, Rosello-Sastre and Martinez-Sanjuan, 1999). Other sources of pain include stretching of the synovium and abnormalities of such periarticular structures as the capsule, ligaments, bursae and medial plica (Papagelopoulos and Sim, 1997).

If one tries to integrate the neural and mechanical theories, one could say that the mechanical component of the problem would provide a “favorable environment”, whilst the neural theory would provide the “precipitating factor” (Sanchis-Alfonso, Rosello-Sastre and Martinez-Sanjuan, 1999).

2.4.5 Aetiology

If it is accepted that PFPS is due to the many causes related to the patella itself or the peripatella structures, and that the symptoms may either be related to changes in the patella articular surface or occur as a result of tracking abnormalities, a number of contributing factors may be isolated (Reid, 1992: 350).

Three major contributing factors that increase the risk of developing PFPS are (Thomee, Augustsson and Karlsson, 1999):

- (1) Over activity,
- (2) Mal-alignment of the lower extremity and/or patella and
- (3) Muscular imbalance of the lower extremity.

Several other predisposing factors will also be discussed.

2.4.5.1 Over activity

Overloading of the patellofemoral joint is the most common cause of PFPS (Witvrouw *et al.*, 2000). Developing PFPS may be related to increasing physical activity and overloading (Juhn, 1999) rather than mal-alignment of the patellofemoral joint (Thomee, Augustsson and Karlsson, 1998).

Fairbank *et al.* (1984) found that patients with anterior knee pain were significantly more involved in competitive sports than age-matched control individuals selected from the normal population. They also found that pain was associated with an increase in activity.

Similar findings were reported by Thomee, Augustsson and Karlsson (1998) but they found that PFPS patients with a high physical activity level did not experience more pain than those with a low activity level. These findings may indicate that increasing physical activity level too drastically might result in an increased risk for developing PFPS (Thomee, Augustsson and Karlsson, 1998).

2.4.5.2 Mal-alignment

2.4.5.2.1 Mal-alignment of the lower extremity

Lower extremity alignment factors associated with PFPS patients include femoral neck anteversion, genu valgum, knee hyperextension, increased Q-angle, tibia varum and excessive rear foot pronation (Thomee, Augustsson and Karlsson, 1998). However, several clinical studies have concluded that the above-mentioned factors, as well as joint mobility, patella position, patient height and weight, were no

different in a normal population when compared to PFPS patients (Reid, 1992: 363-364). Leg length discrepancies are not found to be a significant factor in the aetiology of PFPS (Witvrouw *et al.*, 2000). A high Q-angle may be a contributing factor in maintaining PFPS once it has been acquired (Thomee, Augustsson and Karlsson, 1998).

2.4.5.2.2 Mal-alignment of the patella

Patella shape and position are not associated with the development of PFPS (Reid, 1992: 357). Singerman, Davy and Goldberg (1994) however, state that a high riding patella (patella alta) predisposes to mal-alignment of the extensor mechanism, because the patella is late in engaging the femoral trochlea during knee flexion.

Three different patterns of patella alignment have been described: subluxation without tilting, subluxation with tilting and tilting without subluxation. No difference was found between patients most and least symptomatic when using computerised tomography (Thomee, Augustsson and Karlsson, 1998).

The significance of lower extremity alignment factors and pathological limits need further investigation and the definitions used to define mal-alignment should be re-evaluated. There is little scientific support with regard to determining when alignment is normal, therefore the pathological limits should be clearly defined.

2.4.5.3 Muscular imbalance

Selective dysfunction or insufficiencies of certain components of the quadriceps muscle have been cited as common causes of PFPS (Theil Brody and Theil, 1998). Although other anatomical factors play a role in causing mal-alignment (Sakai *et al.*, 2001), the cause of this mal-alignment in subjects with normal anatomical structure remains controversial and centres around the extent of quadriceps muscle weakness (Powers, Landel and Perry, 1996).

Whether decreased strength is a cause or an effect of PFPS remains undetermined (Thomee, Augustsson and Karlsson, 1998).

Alterations in muscle strength, contraction pattern and flexibility may contribute to PFPS. Insufficient pull of the vastus medialis (VM) may contribute to poor tracking of the patella (Reid, 1992: 361). Vastus medialis oblique (VMO) muscle strength is essential for proper patella tracking (Theil Brody and Theil, 1998). An abnormal relation between VMO and vastus lateralis (VL) muscle activation patterns can disturb the dynamics of the patellofemoral joint (Thomee, Augustsson and Karlsson, 1998).

Powers, Landel and Perry (1996) found no differences in onset or cessation of muscle activity of all vastus muscles during functional activity in subjects without PFPS, when compared to subjects with PFPS. In a review of several studies Thomee, Augustsson and Karlsson (1998) reported contradicting results in terms of the activation of the vastus muscle.

Eccentric torque production during knee extension in PFPS patients, is often poor and the torque curve may be irregular. Both changes have been suggested to represent a deficiency in motor control (Norris, 1998: 242).

Clifton (2003) performed concentric and eccentric isokinetic muscle testing on 20 patients with PFPS. His study confirmed the presence of concentric and eccentric quadriceps muscle weakness, in terms of peak torque values. Thomee, Augustsson and Karlsson (1998) commented similarly on quadriceps muscle weakness. He also noted weakness of the hamstring muscle.

Arthrogenic muscle inhibition appears to play an important role in muscle weakness (Suter *et al.*, 2000) and motor control (Reid, 1992:361-362) in patients with PFPS. Muscle imbalances, with decreased strength of the lower extremity muscles have, been suggested as a potential cause of PFPS (Thomee, Augustsson and Karlsson, 1998).

It is recommended that quadriceps-strengthening exercises accompany treatment for PFPS (Crossley *et al.*, 2001). Exercise treatments emphasize the importance of the VMO because of the medial pull on the patella. However, it is not clear whether it is possible to selectively activate the VMO during exercise (Thomee, Augustsson and Karlsson, 1998). Quadriceps strengthening exercises may simply bring the VMO up to its “threshold” necessary for proper patella tracking, without selective VMO activation (Theil Brody and Theil, 1998).

2.4.5.4 Miscellaneous predisposing factors

A history of trauma and certain gait abnormalities may predispose a person to PFPS (Singerman, Davy and Goldberg, 1994). Reflex sympathetic dystrophy (RSD) may develop following trauma to the knee. RSD is a neurological derangement that usually results from severe or trivial nerve trauma it is not directly related to PFPS. The patient may experience extended periods of exacerbated pain and may be associated with dystrophic and vascular changes. There may be progression to atrophy of affected muscles, skin or bone and loss of joint mobility. Sustained reflex activation of sympathetic nerve fibres is thought to be responsible for the associated symptomatology. (Swenson, 1992:111). If we apply the principles of RSD to PFPS we may hypothesize that following knee trauma the dystrophic and vascular changes associated with RSD may lead to atrophy of the musculature around the knee. A decrease in knee joint mobility and other mobility in the other joints associated with the kinematic chain of the knee may also occur. This may predispose the patient to developing PFPS, as the patella tracking mechanism would be affected.

Genetic predisposition, excessive weight, prolonged synovitis, recurrent haemorrhage into the joint or repetitive intra-articular injection of corticosteroids may play a role in PFPS (Kannus *et al.*, 1999).

Several researchers have reported that third lumbar sclerodermal dysfunction may be a cause of PFPS (Reid, 1992: 351; Wood, 1998).

Salem and Powers (2001) propose that pre- and mid-season fitness levels, sport-specific training experience, hormonal fluctuations, and resistance training techniques may also play a role in causing PFPS.

2.4.6 Presentation

Whereas identification of the underlying pathology is difficult, the classic PFPS picture is easily defined (Reid, 1992: 349). PFPS is often noted as residual pain after an acute knee injury, usually the result of returning to activity before adequate quadriceps strengthening (Garrick and Webb, 1990: 318-319).

Trial *et al.* (1992) notes five groups of people in which PFPS commonly occurs:

- 1- Non-specific anterior knee pain in teenage girls.
- 2- Patella instability in which patella subluxation or dislocation occur.
- 3- Direct trauma to the anterior knee.
- 4- Athletic over-activity.
- 5- Arthritis of the patellofemoral joint.

2.4.6.1 Symptoms

Patients usually present with retropatella or peripatella knee pain. The pain is usually of a dull, aching nature, and occasionally becomes sharp during activities that increase pressure over the kneecap (Davidson, 1993).

Effusions are rare, but a feeling of swelling in the infrapatella fat pad area is frequent. Clicking is a feature with some patients and may be painful or painless (Reid, 1993: 349). After being seated for long periods of time, patients may complain of “locking”. This is not true locking, but rather stiffness resulting in difficulty straightening the knee (Garrick and Webb, 1990: 318).

2.4.6.2 Aggravating factors

According to Powers, Landel and Perry (1996), prolonged sitting, climbing stairs, kneeling and squatting aggravates pain. Reid (1992: 349) adds descending stairs to these points.

Sitting for extended periods of time may also aggravate pain; this is known as the “moviegoer’s” sign (Reid, 1992: 349) or “theatre” sign (Davidson, 1993).

Any activity that involves flexion of the knee will increase the PFJR force and aggravate pain. Activities that aggravate pain are running, cycling and jumping (Kannus *et al.*, 1999).

2.4.6.3 Relieving factors

Rest relieves the pain (Wood, 1998), especially when seated with the knee in the extended position. This is possibly due to a decrease in the PFJR force.

2.4.7 Assessment

2.4.7.1 The History

The main goals of the history are to obtain a specific chief complaint and a thorough outline of activity patterns. Details regarding the onset, nature, pattern and duration of pain are also very important. A slow spontaneous onset, with no or minor injury, suggests overuse or alignment factors. Any blunt injury to the area needs to be recorded. Past therapies especially any surgeries -are important, as are aggravating and relieving factors. (Reid, 1992:365; Wood, 1998).

2.4.7.2 Static alignment assessment

The following alignment factors need to be assessed:

- Pelvic obliquity.
- Patella position.
- Status of genu valgum or varum.
- Foot position (pes planus and pes cavus).
- Hyperextension of the knee (genu recurvatum).
- The relative position of the posterior superior iliac spines.
- The gluteal and calf bulk.
- Camel sign (for a high riding patella).
- Calcaneal position.
- Any leg length inequality.
- Laterally tilted or displaced patella.¹
- The Q-angle. An increase in the Q-angle may be associated with the tendency towards developing PFPS (Sanchis-Alfonso, Rosello-Sastre and Martinez-Sanjuan, 1999). However, not too much emphasis should be placed on the Q-angle, as it is only one of many factors in PFPS and provides no direct correlation with PFPS (Wood, 1998).

2.4.7.3 Specific palpation and stress tests

When pain is originating from the patellofemoral joint, three findings are fairly specific for PFPS (Davidson, 1993):

- (1) Tenderness of the medial and lateral facets on palpation.
- (2) Compression of the patella onto the femoral condyles may produce discomfort.

Both sides of the patella are grasped while the patient contracts the quadriceps muscles; the pressure of the patella against the femoral condyles may cause discomfort.

¹ This test is best performed with the patient seated and having their knee flexed at 90°. Reference a point in the centre of the patella and anterior tibial tuberosity. A line through the transepicondylar axis delineates this line. The normal angle between the delineated lines is 0°, more than 10° are considered abnormal. (Reid, 1992: 365-367; Wood, 1998).

Palpate the structures of the knee and note any tenderness. Rule out effusions of the knee by using the wipe test or ballotment. It is rarely present in PFPS (Reid, 1992: 367).

Test for laxity of the patella by performing the patella apprehension test, as patella instability needs to be identified.

The patella glide test may give an impression of lateral or medial tightness or integrity, while the quadriceps muscles are relaxed. The patella is visualized as being divided into four longitudinal quadrants, and an attempt is made to displace the patella medially:

- A medial glide of less than one quadrant is consistent with tight lateral restraint.
- A glide of one to two quadrants is within normal range; three to four quadrants signify instability. The opposite is true for a lateral patella glide (Wood, 1998).

Range of motion of the knee should be tested passively and then against resistance. The knee should be examined for significant medial and suprapatella plica. Any loss of range of motion, the degree of crepitus and magnitude and location of pain must be recorded. (Reid, 1992:368).

With the patient lying supine, the active compression test (Clark's sign) is performed. The quadriceps should be relaxed. The examiner's web of the hand is placed against the superior pole of the patella and depressed distally. The quadriceps muscle is then contracted. If there is retropatella pain and an inability to hold the quadriceps contraction as the examiner compresses the patella against the condyles of the distal femur, the test is considered positive. (Wood, 1998). The test must be performed bilaterally, repeatedly, and gradually. The involved side should be more painful. (Reid, 1992: 369-370).

Muscle tightness is carefully evaluated at the hip, knee and ankle. Tests for hip flexor tightness (Thomas test), abduction contracture (Ober's test),

hamstrings (straight leg raise), knee flexors (modified Thomas test), soleus and gastrocnemius tightness, are performed. (Reid, 1992:370).

Functional tests like walking, squatting, stair climbing, hopping and duck waddle further assess the magnitude and location of pain (Reid, 1992: 370).

Waldron's test to assesses for the presence of chondromalacia patellae. The anterior part of the knee is palpated whilst the patient performs several deep knee bends. If pain and crepitus occur together during the movement, it is a sign of chondromalacia patellae. (Reid, 1992: 372).

2.4.7.4 Radiological assessment

The combined use of "skyline" projections and measurements derived are important in evaluating contributing causes of PFPS. However, none of these measurements have been used to show a conclusive relationship with regards to the diagnosis or severity of PFPS. The use of lateral projections is valuable in assessing patella alta or baja and patella tendon lengths. (Yochum and Rowe, 1996: 181).

The greatest diagonal dimension from the superior pole to the inferior pole of the patella (patella length) is compared to the distance between the insertion points at the inferior pole of the patella and the notch at the tibial tubercle (tendon length). The two lengths are usually equal. When the patella tendon is more than 20% longer, patella alta is present. Patella baja is present if the patella tendon is more than 20% shorter than the patella. (Yochum and Rowe, 1996: 181).

2.4.8 Diagnosis

The diagnosis of PFPS is currently based on the presence of localized peri- or retropatella pain originating from the peripatella tissue or the patellofemoral joint (Davidson, 1993). Pain must be reproducible by at least two of the following: squatting, stair climbing, kneeling, prolonged sitting or isometric quadriceps femoris muscle contraction (Powers, Landel and Perry, 1996).

The therapist should be able to reproduce the pain that the patient complained of initially. The crux of the diagnosis relies mainly on history and characteristic symptoms (Clifton, 2003).

2.4.9 Management

The etiology of PFPS is still unknown, although many intrinsic and extrinsic factors have been identified. Given the extent of the etiological factors, it follows that many treatment protocols have been advocated (Thomee, Augustsson and Karlsson, 1999).

Managing overuse syndromes involves temporarily reducing or eliminating the aggravating activity. Patients need an individualized treatment plan that takes into account their activity level and lifestyle. (Austermuhle, 2001).

The medical diagnosis of PFPS is generally uncomplicated, but the diagnosis made by physical therapists is more complex. The impairments, functional limitations and disabilities found during examination guide therapeutic interventions. Once an accurate diagnosis has been made, interventions that address specific components of the patients' problem (i.e. Pain, muscle imbalance, poor flexibility and mal-alignment) can be initiated. (Thein Brody and Thein, 1998; Juhn, 1999). Patient compliance is an important factor in the treatment of PFPS (Thomee, Augustsson and Karlsson, 1999).

In a systematic review of the literature regarding non-operative treatments for PFPS, Arroll *et al.* (1997) found only 5 randomized clinical trials and concluded that there is little evidence on which to base therapy. In a similar review, Crossley *et al.* (2001) suggested that clinicians should institute a program of education, stretching and quadriceps strengthening (including eccentric exercises) and possibly make use of soft corrective foot orthoses. They neither promoted nor refuted the addition of patella taping, patellofemoral orthoses, low-level laser therapy, manipulations or mobilizations, or acupuncture for the conservative management of PFPS.

Appropriate utilization of the interventions is dependant upon patient symptomology as it relates to the stage of healing. In the acute stage, the immediate goals of treatment should be to decrease pain and inflammation and loading at the patellofemoral joint. Treatments at this stage include cryotherapy, rest and nonsteroidal anti-inflammatory medication. (Reid, 1992: 13-20). Once acute symptoms have passed, the intermediate goals should be to focus on restoring soft tissue mobility and force production ability in the prepatella tissue. Treatments at this stage include isokinetic exercises and stretching. Adjunctive supports, such as taping or orthotics, may be helpful. (Thein Brody and Thein, 1998). The late phase treatment focuses on returning the patient to their previous level of activity and instructing them on long-term care for the knee. This may include stretching, mobilization, strength training, activity modification and the use of ice after activity, with the return to activity requiring a functional activity progression programme. This requires a series of planned progressive functional activities that lead to the previous level of activity. (Thein Brody and Thein, 1998). Once the patient's symptoms have decreased, they should gradually increase their physical activity. For example: a runner should start with walking or jogging, progress to running at half speed in a straight line and eventually running at full speed with cutting and hill work. (Davidson, 1993).

2.4.9.1 Manipulative therapy

There is little published work documenting spinal and pelvic manipulation as part of the treatment protocol for PFPS (Wood, 1998).

In a single subject experiment, Meyer *at al.* (1990) reported good results with long axis manipulation of the tibio-femoral joint and patella mobilizations for the treatment of PFPS. Ultrasound therapy also formed part of the subjects' treatment and hence no valid comment can be made regarding the effect of manipulating the tibio-femoral joint and mobilization of the patella in PFPS patients.

In another single subject experiment, benefits with manipulation of the lumbar spine, sacroiliac joints, pubic symphysis and femoroacetabular joints were observed in patients with PFPS (Wood, 1998). Multiple interventions were used during these single subject experiments and hence no valid conclusions, regarding the effect of manipulative therapy on PFPS, may be drawn.

Rowlands and Brantingham (1999) showed that patella mobilization did not statistically improve the symptoms of PFPS patients when compared to a placebo and Stakes (2000) showed no statistical improvement when combining lumbar spinal manipulation and patella mobilization for the treatment of PFPS.

The interdependence of the lumbo-pelvic region with the lower extremity kinetic chain has been noted. An example of this is an anterior tilt of the pelvis, which leads to internal rotation of the femur, which is a source of PFPS. Similarly, hyperpronation of the foot can cause joint dysfunctions in the lumbar and sacroiliac regions. Therefore intersegmental motion restrictions may restrict pelvic motion and translate abnormal forces to the knee. With the correction of postural asymmetries and lumbopelvic dyskinesias² there is a decrease in torsion of the femur and normalization of factors that influence

² A group of involuntary movements that appear to be a fragmentation of the normal smooth controlled movement (Oxford concise medical dictionary, 1998: 199).

patellofemoral biomechanics. (Walters, 1993: 155). Therefore it is likely that the reported success of manipulative therapy in the treatment of PFPS results from the restoration of alignment and lumbopelvic kinematics, as well as the relief of reflex sympathetic dystrophy (RSD). This hypothesis has however not been adequately addressed in the literature. (Wood, 1998).

2.4.10 Prognosis

A reasonable outcome can be expected after 6 months of conservative treatment (Jackson, 2001). In a 7 year follow-up, on PFPS Kannus *et al.* (1999) reported that 76% of their patients had recovered by 6 months and that the results hardly changed' after 7 years.

Many patients with PFPS will however experience a return of their symptoms at some point in time (Theil Brody and Theil, 1998). Patient education is the most important factor in improving the prognosis for PFPS (Clark *et al.*, 2000), as an educated patient will be able to prevent a significant exacerbation of symptoms (Theil Brody and Theil, 1998).

2.5 JOINT DYSFUNCTION

2.5.1 Definition

The term dysfunction implies that at one anatomical level, the components of the joint are not functioning "normally" (Kirkaldy-Willis and Burton, 1992: 105). The words subluxation and fixation are commonly used to describe this state of altered functioning or dysfunction. The term fixation describes a specific type of dysfunction that is applied to a functional spinal unit that is restricted in any one or all six degrees of freedom. (Lopes, 1992:55).

According to Haldeman (1992: 623) a fixation is a state whereby an articulation has become temporarily immobilised in a position that it may normally occupy during any phase of physiological movement. Haldeman (1992: 627) adds that a subluxation is an aberrant relationship between adjacent articular structures that may have functional or pathological sequelae, causing an alteration in the biomechanics and/or neurophysiological reflex of these articular structures. Joint fixation, or loss of motion, is only part of the subluxation complex or dysfunctional joint. Other signs and symptoms, adapted from Kirkaldy-Willis and Burton's (1992: 109) model for low back are:

- Symptoms: local or referred pain and painful movement.
- Signs: local tenderness, muscle spasm, a normal neurological exam and, as mentioned above, hypomobility.

2.5.2 Causes of joint dysfunction

The causes of joint dysfunction can be singular or multifactorial. Several mechanisms may occur together in a single case or could predispose one to be affected by another (Lopes, 1992:56).

Noxious impulses from nociceptive stimulation of free nerve endings in an area of chemical or mechanical irritation may be a primary etiological factor in

the vertebral subluxation complex (Lopes, 1992:65). Conversely, one can also argue that dysfunction adversely affects nerve endings, causing inhibition of nerve function (Wyke, 1985: 72-77).

The SI joint has been shown to be a source of lower back pain (Sakamoto *et al.*, 2001). The pain is thought to be due to nociceptive stimulation around the SI joint. SI joint fixations are thought to be responsible for pain associated with lower back pain originating from the SI joint (Cassidy and Mierau, 1992: 218). SI joint dysfunctions have been associated with PFPS (Suter *et al.*, 1999; Suter *et al.*, 2000).

Other possible mechanisms of fixation include muscle spasm, meniscoid entrapment, articular adhesions and edema and disc derangement. Contracture of ligaments, muscles and tendons may also resist intersegmental motion (Lopes, 1992:56; Leach, 1994: 44-47).

2.5.3 The neurobiological effects of manipulation

The following reflex mechanisms and structures in and around the joint have been linked to muscle spasm and the reduction thereof: articular receptors, the sympathetic nervous system, facilitated motor neuron pools, Golgi tendon organs and muscle spindles (Gatterman, 1990: 44; Bergmann, 1995: 110-111).

Following manipulation, presynaptic nociceptive inhibition of proprioceptors occurs (Lopes, 1992:66). Manipulation has been hypothesized to produce significant short-term bursts of proprioceptive transmission in the larger-calibre myelinated alpha afferent fibres arising from the joint capsules, ligaments and in the muscle spindles of the local paraspinal musculature. These larger fibres signals are believed to modulate the interneuronal pool via the dorsal spinal root ganglion and the substantia gelatinosa subsequently close the gate on pain transmission. (Dhami and DeBoer, 1992: 121). Nociceptive reflexes from an irritated area can -through complex mechanisms-

affect the sympathetic nervous system, which increases the muscular tone of this area. Thus, by interrupting or decreasing the nociceptive input to the central nervous system, the normal muscle tone could be reset (Colloca 1997: 42).

In a similar manner, stretching of the joint capsule and the subsequent stimulating of mechanoreceptors, is said to reflexly inhibit facilitated motor neuron pools that are responsible for the increased muscle tone and spasm that are found with joint dysfunction (Kirkaldy-Willis and Burton, 1992: 288, Colloca, 1997: 47).

When articular surfaces are separated during manipulation, the hypertonic intersegmental muscle is suddenly stretched, initiating muscle spindle mediated reflexes that relieve the hypertonicity (Leach, 1994: 44).

Golgi tendon organs act as brakes and limit excessive joint movement by initiating reflex inhibition of motor activity in the muscles operating the joint (Gatterman, 1990: 44). A high velocity thrust is thought to be able to stimulate golgi tendon organs around a joint, causing reflex inhibition of motor activity and thus breaking into the muscle spasm cycle (Suter *et al.*, 2000).

2.5.4 Sacroiliac joint dysfunction

Forward bending, twisting and lifting are most likely the activities responsible for strains in this joint (Cassidy and Mierau, 1992:216). Sacroiliac (SI) syndrome or symptomatic SI joint are interchangeable terms used to describe the presence of pain and joint dysfunction in the SI joint (Cassidy and Mierau, 1992:218-219; Suter *et al.*, 2000)

Hyper-tonicity in the muscles associated with SI joint movements, leads to a functional dyskinesia of the pelvis. A fixation develops in the SI joint and results in a shift in the axis of rotation, leading to the development of a subluxation complex (Walters, 1992: 155).

SI pain may be characterized by lower back pain plus pain and tenderness over the SI joint. This pain may extend to the buttock, groin, genitalia, trochanteric region, thigh, heel and to the lateral foot. Pain in the SI joint is associated with a marked decrease in mobility of the joint. A diagnosis of SI syndrome can be made when pain and motion fixation of the SI joint are present in the absence of neurological signs (Cassidy and Mierau, 1992: 216-218; Walters, 1993:155+161).

Suter *et al.* (2000) described a symptomatic SI joint as exacerbated pain over the posterior superior iliac spine (PSIS) when provocation tests such as; Yeoman's / Erichsons, Patrick's Faber and sacral compression, were performed and found to be positive with pain over the SI joint. In a study on 28 subjects suffering from patellofemoral pain syndrome, Suter *et al.* (2000) found that every subject had either a symptomatic or an asymptomatic SI joint dysfunction.

Restrictions in the SI joint occur in either the upper or lower joint. These areas may be restricted in either flexion or extension. The standing flexed-knee-raising test is a popular test used to identify any restrictions present in the SI joint (Schafer and Faye, 1990: 260-283). Although this test appears to be reliable, its validity remains to be proven (Cassidy and Mierau, 1992: 220).

Manipulation is recommended as the first line of treatment for SI syndrome. However, successful treatment through manipulation does not however prevent recurrence of the problem. (Cassidy and Mierau, 1992: 221). There are many techniques to manipulate the SI joint. However, Cassidy and Mierau (1992: 221) find the side-posture method to be the best.

2.5.5 Tibiofemoral dysfunction

Knee joint dysfunction has received little attention in the literature. The normal biomechanics of the knee are frequently altered by fixations that may be the cause or the effect of pathological changes. Without a normal degree of movement in the knee, voluntary knee joint movements are impaired and painful. (Schafer and Faye, 1990:385-386).

Any of the following movements may become restricted in the tibiofemoral joint: medial or lateral side tilts, anteroposterior / posteroanterior glide, knee rotation (lateral or medial) and long axis distraction. The restriction is removed by applying a short dynamic thrust at the end of the movement tested and found to lack a particular movement. (Schafer and Faye, 1990:393-396).

2.6 ARTHROGENIC MUSCLE INHIBITION

2.6.1 Definition

Knee joint pathologies, in general, are associated with a loss of knee-extensor muscle strength. This weakness has been attributed to arthrogenic muscle inhibition (AMI) (Suter *et al.*, 2000).

AMI is a presynaptic, ongoing reflex inhibition of musculature surrounding a joint following distension or damage to structures of that joint. AMI is a natural response designed to protect the joint from further damage (Hopkins and Ingersoll, 2000).

In some cases, the response may be an arthrogenic muscle response (rather than inhibition). Experimental effusions of the ankle have been shown to cause facilitation of the muscles of the leg (This is defined as the area between the ankle and the knee.). The resulting muscle spasm is thought to help protect the ankle from further injury. Without other means of protecting a joint, AMI is the body's method of choice to protect injured extremities. (Ingersoll, Palmieri and Hopkins, 2003).

2.6.2 Aetiology

Pain and disuse are often blamed for the inhibition and muscle atrophy following joint injury (Helliwell and Jackson, 1994). Studies using experimental infusions have shown that AMI can occur independently of the pain sensation (Young, 1993). AMI has been identified in subjects with both symptomatic and asymptomatic osteoarthritis (Arokoski *et al.*, 2002). Although AMI can occur independently of the conscious appreciation of pain, the possibility that afferent impulses from nociceptors may be an important contributing factor even if it does not rise to consciousness cannot be excluded (Young, 1993).

Due to the observation that damage to the joint receptors results in impairment of proprioception and that the extensor musculature of the upper thigh is reduced following knee trauma (to an extent which exceeds inactivity atrophy), the joint receptors have been assigned great importance in AMI (Freiwald, Reuter and Engelhardt, 1999).

Free nerve endings and specialized nociceptors may play a role in inhibition, but the primary effect seems to stem from mechanoreceptor activity (Hopkins and Ingersoll, 2000). AMI therefore results from the activity of many different mechanoreceptors within a joint, namely: Ruffini endings, Golgi-like endings and Pacinian corpuscles (Hopkins and Ingersoll, 2000) and free nerve endings may also play a role in AMI (Ingersoll, Palmieri and Hopkins, 2003; Freiwald, Reuter and Engelhardt, 1999).

2.6.3 Natural history

If arthrogenic muscle inhibition (AMI) is not resolved after the initial injury has occurred, mechanoreceptor activity will continue to alter muscle activity surrounding the joint. The patient will replace normal muscle patterning with an adapted functional motor program. (Ingersoll, Palmieri and Hopkins, 2003).

Ongoing inhibition of a muscle, with its concomitant decrease in physical activity, can have numerous long-term effects on a number of tissues in the body. In muscle, there may be type I fibre atrophy, decreased cross-sectional area, and decreased oxidative enzyme activity. Periosteal and subperiosteal resorption, decreased strength and stiffness, diminished load to failure and energy storage capacity may occur in bone. Ligaments may lose tensile strength, experience decreased load to failure, become more elongated, and be less stiff. Negative neural factors include depolarized muscle fibre membranes, decreased potential at motor end plates, and reduced Na⁺ - K⁺ transport across membranes. Removal of muscle inhibition, along with

maintained activity levels, could eliminate or reduce these negative effects. (Ingersoll, Palmieri and Hopkins, 2003).

2.6.4 Pathophysiology

The spinal cord consists of a complex system of channels relaying information from several parts of the body. The central and peripheral nervous systems work together to gather, transmit, and process information from many different neurophysiological systems in order to coordinate movement. Joint movement provides supraspinal centres with constant information about the environment, position, and movement. Changes in afferent input to the spinal cord from the joint appear to be the most influential factor associated with AMI. (Hopkins and Ingersoll, 2000).

Abnormal afferent information from mechano- and nociceptors surrounding the damaged joint, act on inhibitory interneurons, which synapse on the motor neuron (MN) pool of the joint musculature across that joint (Hurley and Newham, 1993). The information from inhibitory interneurons decreases the ability of recruitment within the MN pool, and therefore decreases the force of any contraction stemming from that MN pool (Hurley and Newham 1993; Hopkins and Ingersoll, 2000).

Post knee surgery assessment of the peripheral afferent and efferent pathways, proximal segments of the peripheral nerves, and central processing of information, has revealed that there are no deficits in any of the above mentioned components of the peripheral and central nervous systems (Freiwald, Reuter and Engelhard, 1999).

Lesions to peripheral mechanoreceptors functionally modify central somatosensory pathways. The cortex is involved in complex spatial integration of articular proprioceptive inputs. Measurable changes in muscle strength and muscle atrophy may be due to altered central nervous system under-activation of the involved musculature (Freiwald, Reuter and Engelhard,

1999). Some authors have however suggested that joint afferents do not change cortex activity (Hopkins and Ingersoll, 2000).

Clinical observation suggests that the vastus medialis is the most affected quadriceps muscle from AMI. Some authors have observed that the vastus medialis was inhibited with less fluid than any of the other quadriceps muscles during artificial effusion (Stokes and Young, 1984; Freiwald, Reuter and Engelhard, 1999; Hopkins and Ingersoll, 1999). Recruitment patterns were also noted to have changed in the quadriceps muscle with patellofemoral dysfunction (Freiwald, Reuter and Engelhard, 1999). Selective muscle fibre type inhibition has been questioned in the literature (Freiwald, Reuter and Engelhard, 1999). There is no evidence that any specific muscle fibre type is inhibited more than another (Hopkins and Ingersoll, 2000).

2.6.5 Types of inhibition

2.6.5.1 Postsynaptic

Inhibition in the nervous system is either postsynaptic or presynaptic. Synapses between neurons or between a neuron and membrane may be either excitatory or inhibitory. Both excitatory and inhibitory processes result in the release of a neurotransmitter at the terminal end plate. If the neurotransmitter is inhibitory, then postsynaptic inhibition occurs. (Solomon, Schmidt and Adragna, 1990: 423-424).

2.6.5.2 Presynaptic

Presynaptic inhibition is generally caused by a decrease in neurotransmitter release from the presynaptic terminal. The purpose of presynaptic inhibition is to decrease the effectiveness of just one type of neuron synapsing on the membrane, and may be more specific than postsynaptic inhibition (Hopkins and Ingersoll, 2000).

Arthrogenic muscle inhibition (AMI) is likely a combination of presynaptic and postsynaptic inhibition. Afferent supply to the interneuron is mediated by

postsynaptic neurotransmitters. The information travelling from the interneuron to the motor neuron (MN) pool is then presynaptically mediated (Hopkins and Ingersoll, 2000).

2.6.5.3 Renshaw inhibition

Other inhibitory processes may also play a role in AMI. Recurrent inhibition is inhibition mediated by Renshaw cells found on the efferent loop near the alpha MN pool. The Renshaw cell is excited by alpha MN activity, which then inhibits the interneuron, projecting to its synergists. A recurrent inhibition cycle is set up, where there is inhibition of the synergists supplied by that MN pool and disinhibition of the antagonists. Renshaw cells are under central control, receiving information through descending fibres from the brain stem and cortical pathways, helping to modify the effect of these cells. (Hopkins and Ingersoll, 2000).

2.6.5.4 *Reciprocal inhibition*

Reciprocal inhibition is caused by type I alpha (Ia) inhibitory interneuron activity. Muscle spindle primary fibres respond to stretch, resulting in afferent activity that synapses on interneurons (one of these interneurons is the Ia inhibitory interneuron). Stimulation of this neuron results in inhibition of the antagonist muscle and its synergists (Hopkins and Ingersoll, 2000). Also involved in this loop is the gamma MN system. This system is important in functional regulation of Ia receptors in the muscle during contraction. Hopkins and Ingersoll (2000) suggest that stimulation of gamma MNs by joint afferents helps contribute to continuous inhibition.

2.6.6 Structures related to arthrogenic muscle inhibition in the quadriceps

It has been suggested that inhibitory stimuli from the knee joint are responsible for arthrogenic muscle inhibition (AMI) in the quadriceps muscle group. Evidence in support of this idea comes from studies that experimentally induced quadriceps AMI, by injecting saline into the knee joint (Iles *et al.*, 1990; Hopkins *et al.*, 2001).

Structures within the knee joint that have been shown to induce quadriceps AMI are the lateral and medial collateral ligaments (Kim *et al.*, 1995), the anterior and posterior cruciate ligaments (Fisher-Rasmussen *et al.*, 2002; Lewek *et al.*, 2002), the joint capsule (Hopkins *et al.*, 2001), perimeniscal tissue and the outer third of the menisci (Hopkins and Ingersoll, 2000). The lateral retinaculum has also been identified as a possible source of AMI, although it is not known whether it plays a significant role (Hopkins and Ingersoll, 2000; Sanhis-Alfonso and Rosello-Sastre, 2000). Immobilization and effusions of the knee are also associated with quadriceps AMI (Cruz-Martinez, Ramirez, and Arpa, 2000; Hopkins and Ingersoll, 2000; Hopkins *et al.*, 2001).

The sacroiliac (SI) joint has been linked to AMI within the quadriceps muscle group (Suter *et al.*, 1999; Suter *et al.*, 2000). Sakamoto *et al.* (2001) showed that the SI joint possessed mostly nociceptive mechanoreceptors, stimulation of which, can give rise to inhibition of the innervated motor neuron pool. The SI joint is innervated by the anterior primary divisions of L2-S2, which project on to the main lower limb nerves (Suter *et al.*, 2000). Suter *et al.* (2000) found that all subjects in their study (diagnosed with anterior knee pain) had a SI joint dysfunction that was either symptomatic or asymptomatic on the ipsilateral side of the pathological knee.

2.6.7 Measuring arthrogenic muscle inhibition

Arthrogenic muscle inhibition (AMI) is simply a reduction in MN pool recruitment and this may be measured indirectly by taking into account changes in recruitment. It may include voluntary motor unit recruitment as measured by a dynamometer or through electromyography. It might also include involuntary measures of MN recruitment through controlled stimulation of sensory fibres and evaluation of the reflexive twitch contraction using the Hoffmann reflex. It may also be measured combining a voluntary contraction with a superimposed electrical impulse (eg, interpolated twitch techniques). Each method has advantages and disadvantages. (Ingersoll, Palmieri and Hopkins, 2003).

2.6.7.1 Voluntary Force Measurement

Measuring voluntary force output of a MN pool is a simple measure that may be performed with little equipment. Decreased voluntary contraction is one of the final outcomes of AMI. The difference in a baseline maximum voluntary contraction (MVC) and an MVC following injury is essentially inhibition. (Hopkins and Ingersoll, 2000).

Comparison of force measurements of the injured leg to the contralateral or uninjured leg has been used to measure the amount of inhibition present (Stokes and Young, 1984). However crossed spinal pathways may transmit information to the uninvolved leg, which may inhibit the joint musculature of the contralateral leg. Therefore, this method of comparison may not be totally acceptable as a valid comparison (Hopkins and Ingersoll, 2000).

Muscle force comparisons are very simple to perform. However, these measurements also have some drawbacks. In order to effectively measure differences in voluntary force production, the subject must be willing and able to perform a MVC. If a subject is asked to perform a voluntary contraction post-injury, there are psychological factors such as perceived pain and lack of confidence that could hinder his or her ability to perform an MVC. This

measurement method also uses an entire group of muscles with aid from synergists that may or may not be inhibited. (Ingersoll, Palmieri and Hopkins, 2003). As it is impossible to measure independent muscles separately and as the contralateral leg comparison is not valid in establishing a baseline measurement, the interpolated twitch technique may be used as a comparison to the pathological measurement (Hopkins and Ingersoll, 2000).

The interpolated twitch technique is a combination of a MVC and an additional supramaximal external stimulus to make up for the inhibited portion of the MN pool. This technique allows for a measurement of AMI without a baseline torque measurement. However the validity of this measurement is questionable as the magnitude of the force generated during a twitch is very small compared with the background force, and it can easily go undetected (Hopkins and Ingersoll, 2000).

2.6.7.2 The Hoffmann Reflex

The Hoffmann reflex (H-reflex) is an indirect measure of motor neuron (MN) pool recruitment. By assessing the twitch response of a muscle created by stimulating a mixed nerve, the amount of MN pool activity can be established. Surface electromyography is used to visualize the muscle activity (Ingersoll, Palmieri and Hopkins, 2003).

Electrical stimulation of a mixed nerve evokes two distinct electromyographic responses from the affected muscle. One response to the stimulus is an action potential resulting from primary afferent type I alpha stimulation, which in turn excites alpha MN's in the anterior horn of the spinal cord. This response is the H-reflex. As the intensity of the stimulus increases, more afferent fibres are stimulated, causing more MN's to be recruited within the MN pool. This is represented as an increased amplitude of the twitch of the affected muscle. As the external stimulus intensity is increased a second response appears. This response is a direct stimulation of efferent alpha MN fibres, and it is termed the M-response (Hopkins and Ingersoll, 2000).

The amplitude of the H-reflex represents the portion of the MN pool that was stimulated from afferent activity. Inhibition results in a decrease in MN pool excitability hence a decrease in the H-reflex amplitude. It can be very difficult to obtain reliable H-reflex measurements, as H-reflex responses may vary with such things as head and body posture, foot position, eye movement, and remote muscle contractions.

The H-reflex is a very sensitive measurement that requires great control. It is a measure that may be performed while the subject is resting, with no voluntary effort required, which is extremely useful when examining a pathological population. It is also a very sensitive measure that may be used to detect small changes in MN pool recruitment. A single muscle may also be investigated as opposed to a large muscle group, which may allow for investigation of selective inhibition of certain muscles in a muscle group. (Hopkins and Ingersoll, 2000). The H-reflex however, does not however take into consideration supraspinal inputs that may affect the MN pool during voluntary exercise (Ingersoll, Palmieri and Hopkins, 2003)

2.6.8 Treatment

Arthrogenic muscle inhibition (AMI) can be eliminated or diminished by removing, masking, overriding or otherwise altering inhibitory interneuron activity. Anything that may alter, slow or compete with mechanoreceptor feedback may be a candidate. These include pharmacological agents, cryotherapy, transcutaneous nerve stimulation and manipulative therapy. (Ingersoll, Palmieri and Hopkins, 2003).

2.6.8.1 Manipulative therapy

Manipulative therapy has been shown to be effective in reducing quadriceps muscle inhibition (MI). Suter *et al.* (2000) showed -using a randomized, double blinded, controlled clinical trial- that sacroiliac joint (SI) manipulation was effective in reducing MI and increasing the strength of the quadriceps muscle

group. The inverse relationship of muscle strength and MI did not however reach statistically significant levels. Yet, in their earlier study, they had shown that this relationship did reach a statistically significant level (Suter *et al.*, 1999). Although the researchers tried to test a homogenous group, the homogeneity of their sample (in both trials) was compromised by the inclusion of some subjects who had previously undergone knee surgery.

Manipulation of a joint has been proposed to activate mechanoreceptors from structures in and around the manipulated joint. The altered afferent input arising from the stimulation of these receptors is thought to cause changes in the motor neuron excitability, with a subsequent decrease in arthrogenic muscle inhibition (William, 1997: 144; Suter *et al.*, 2000).

2.7 ISOKINETIC DYNAMOMETRY

2.7.1 Isokinetic exercise

The term isokinetic exercise refers to a process in which a body segment accelerates to achieve a pre-selected fixed speed with totally accommodating resistance throughout the range of motion. The amount of force exerted by the subject is always matched by that of the machine, therefore the subject can never exceed the speed no matter how much effort they exert. As a result, isokinetics has the capability to load a muscle maximally throughout the entire range of motion. (Cybex, 1996:1-9). This makes isokinetic exercise a safer and more efficient form of exercise than isotonic exercise, which maximally loads the muscle at its weakest points. Additionally, when the patient begins to fatigue and is unable to continue with the exercise, they are still loaded maximally with the isotonic resistance. Isokinetic resistance will accommodate to this variable. (<http://www.biodex.com>).

Two types of isokinetic contractions are possible: concentric or eccentric contractions (Cybex, 1996:1-9).

2.7.1.1 Concentric contractions

Concentric contractions are defined as the development of tension by a muscle while the origin and insertion approximate each other. This involves the shortening of the muscle fibres with the origin and insertion approximating (Cybex, 1996:1-9). This is also referred to as positive work by Davies (1992: 25).

2.7.1.2 Eccentric contractions

The development of tension that occurs as the origin and insertion move away from each other defines an eccentric contraction. This involves the lengthening of muscle fibres with the origin and insertion separating (Cybex, 1996:1-9). Davies (1992: 25) refers to this as negative muscle work.

2.7.2 The use of isokinetics

Clinical testing is one of the most important aspects of dynamometry. The primary function of an isokinetic dynamometer is to provide some form of quantitative measure of muscular force of a limb at any given moment or position. Isokinetic testing provides an effective way of attaining objective measures. Isokinetic dynamometry has also been used in injury rehabilitation and muscle training. (Deans, 2001).

The following list represents measurable values and their definitions:

- Peak torque: Defined as the highest muscular torque produced by the muscle. Peak torque indicates the muscle's maximum strength capability. It can be evaluated specific to time (Torque at .20sec) or to range of motion (Torque at 30°). Peak torque is an absolute value. When used alone it is difficult to assess the specific strength of a person.
- Angle of peak torque: Is defined as the point in the range of motion (ROM) where peak torque is produced. It usually occurs at the same range in the ROM for similar movements and speeds and typically occurs in the mid range of a motion. This should be at the point in the ROM where the length-tension relationship of the muscle is maximal.

[All definitions adapted from: Marrule, 1996: 110-114]

There is little scientific evidence regarding the reliability and validity of these values. The peak torque value is the only value that is both valid and reliable. This reduces the clinical application of several of these performance measures, thus limiting the utilization of isokinetic dynamometry as an effective measurement device (Deans, 2001).

2.7.3 Advantages and limitations of isokinetic testing

2.7.3.1 Advantages of isokinetic testing

- Permits isolation of muscle groups.
- Provides accommodating resistance to maximal exercise throughout the range of motion.
- Presents quantifiable data for peak torque, work and power.
- (Perrin, 1993:7)
- Reduces the chance of overload injury.
- Accommodates for pain fatigue.
- Full range of speed for testing and exercise (within limits of the machinery).
- Provides reproducible measurements. [Cybex, 1996: 1-10]

2.7.3.2 Limitations of isokinetic exercise

- Non-specific functional training for the lower extremity in the closed kinetic chain.
- Angular velocity movements do not approach functional speeds. [Perrin, 1993: 7]

There are numerous variables that may affect the reliability and validity of the measures obtained during isokinetic testing. To ensure reproducibility of results, some guidelines have been established. The guidelines consist of the following parameters:

- Patient education. It is important to familiarize the subject with the testing procedure. The use of practise sessions and patient comfort has been shown to increase the maximum voluntary effort of the subject, but may result in a “learning effect”.
- The testing order should always remain the same.
- The axis of rotation used. Any unnatural joint movements should be avoided.

- Warming the muscle up before testing will ensure a safer and more effective muscle contraction.
- Stabilization- to prevent excessive movements during testing.
- Angular velocities used during testing. The angle at which the tested limb moves through its' range of motion. The tester therefore presets this value.
- Calibration of the equipment used.
- Gravity correction of the limb being tested. The gravity effect torque is the torque produced by the tested limb and attachment. Gravity correction allows for the measurement of the torque value of the tested limb only.
- The number of test repetitions performed.

[Adapted from: <http://www.biodex.com>]

- Patient positioning. Evidence indicates the importance of postural specificity when conducting clinical testing. Due to the elastic characteristics of muscles, any change in the length of the muscle as a result of active or passive joint motion, will incur an associated change in the tensile strength of that muscle. Keeping the hip angle constant is very important during testing of the lower limb (Deans, 2001).
- The use of visual feedback and verbal encouragement has been shown to increase the maximum voluntary force output (Campenella, Mattacola and Kimura, 2000).

Patella compression forces are almost 12 times higher than walking and 6 times higher than running at the functional range of motion during isokinetic knee extension testing. For this reason, some authors argue that patients with PFPS should only perform submaximal efforts, which is at odds with the rationale of the testing procedure, which requires maximal contractions (Nisell and Ericson, 1999).

Notwithstanding the limitations of isokinetic testing, if bilateral or reciprocal muscle group comparison is required, then isokinetic dynamometry should be

the modality of choice for measurement (Pincivero, Lephart and Karunakara, 1997).

2.7.4 Contraindications to isokinetic sessions

2.7.4.1 Absolute contraindications

- Soft tissue healing constraints.
- Severe pain.
- Extremely limited range of motion.
- Severe joint effusion.
- Unstable joint.
- Acute sprain.

2.7.4.2 Relative contraindications

- Pain.
- Limited range of motion.
- Effusion due to synovitis.
- Chronic third degree sprain.
- Pregnancy.

[Adapted from Cybex, 1996: 1-13]

2.7.5 Data analysis

When using isokinetic dynamometry as a measuring tool many values can be assessed in relation to each other. Not one value is able to accurately quantify muscular function. The agonist to antagonist ratio was used in this study.

2.7.5.1 Agonist to antagonist ratio

This represents the reciprocal muscle group ratio (ie. peak torque hamstring: peak torque quadriceps). An imbalance in this ratio may predispose a joint to injury as opposing muscle groups provide dynamic joint stability (www.biodex.com). The normal Quadriceps: Hamstring ratio is ranges from

50% to 80%, in favour of the quadriceps. This ratio is changes as the testing speed is altered. (Rosene, Fogarty and Mahaffey, 2001).

Curve analysis is also useful in interpreting muscle pathology (Clifton, 2003).

2.7.6 Conclusion

Reproducibility and reliability of isokinetic testing for a desired protocol should be sufficient enough so that training or injury- induced changes in muscle strength are not attributed to instrument or testing error. The ability to quantify reliable and relatively precise values for maximal strength and endurance, as measured by isokinetic dynamometry, should provide a valuable tool for the evaluation of muscular capability and injury assessment (Pincivero, Lephart and Karunakara, 1997).

2.8 SUMMARY

The reviewed literature indicates the need to further investigate any treatments that are aimed at reducing or eliminating arthrogenic muscle inhibition (Ingersoll, Palmieri and Hopkins, 2003).

Knee joint pathologies, in general, are associated with a loss of knee-extensor muscle strength. This weakness has been attributed to arthrogenic muscle inhibition (AMI). (Suter *et al.*, 2000). Both the knee and sacroiliac joints have been linked to AMI in the quadriceps muscle group (Iles, Stokes and Young, 1990; Suter *et al.*, 1999; Suter *et al.*, 2000; Hopkins and Ingersoll, 2000). Mechanoreceptor stimulation plays an important role in AMI and anything that may alter, slow or compete with mechanoreceptor feedback may affect AMI (Ingersoll, Palmieri and Hopkins, 2003).

A decreased voluntary contraction is one of the final outcomes of AMI. Measuring the voluntary force output of a motor neuron pool (ie: peak torque) is a simple measure that may be performed with little equipment. (Hopkins and Ingersoll, 2000).

The use of isokinetic dynamometry has been shown to be a valuable tool for assessment and evaluation of muscular function and pathology and is at present considered the most appropriate indicator of functional status in subjects with PFPS (Pincivero, Lephart and Karunakara, 1997).

Therefore this research aims to determine if manipulation of the knee (tibio-femoral) joint is as effective as SI joint manipulation in increasing quadriceps muscle strength in PFPS patients, using isokinetic dynamometry to quantify the muscle strength. This study will also investigate the effect of combining manipulative therapy of the tibio-femoral and SI joints, on quadriceps muscle strength in PFPS patients.

CHAPTER THREE

METHODOLOGY

3.1 INTRODUCTION

Reproducibility of results is a requirement of modern science. It is therefore important to describe the process involved in this study, so that it can be repeated. This chapter focuses on the design of the study, the sampling procedure, the interventions that were applied and the data collected, as well as the statistical analysis applied to that data.

3.2 OBJECTIVE OF THE STUDY

This pilot study proposed to evaluate the effect of three manipulative treatment protocols on quadriceps muscle strength in individuals with Patellofemoral Pain Syndrome (PFPS), in terms of isometric contractibility and levels of perceived pain.

Statistical analysis of the data obtained investigated the relationship between quadriceps muscle strength and levels of perceived pain in individuals with PFPS.

3.3 STUDY DESIGN

This pilot study was a prospective clinical trial.

3.3.1 The sampling procedure

This study was limited to patients with Patellofemoral Pain Syndrome (PFPS), in order to obtain a uniform sample group. A convenience, purposive sampling approach was adopted in this study. Subjects were recruited by placing advertisements in local newspapers, as well as around the Durban Institute of Technology campus and running clubs (see Appendix A). The research program was explained to respondents and those who did not meet the criteria for the study were excluded. These criteria were as follows:

3.3.1.1 Inclusion criteria

- ❖ Studies have shown that persons with radiographic knee osteoarthritis (OA), whether symptomatic or not, have weaker quadriceps muscle strength than those without knee OA. This muscle weakness has been attributed to arthrogenic muscle weakness (Arokoski *et al.*, 2002). Brandt (2002) stated that little radiographic evidence of OA exists in people under the age of 45 years. Subjects were between the ages of 18-45 in order to recruit patients whose quadriceps muscle strength was affected by AMI as a result of PFPS and not OA of the knee.

Subjects needed to have:

- ❖ Localized peri- or retro-patellar pain originating from the peri-patella tissue or the patellofemoral joint (Rowlands and Brantingham, 1999).

The peri- or retro-patellar pain needed to be reproducible with at least two of the following:

- Squatting
 - Stair climbing
 - Kneeling
 - Prolonged sitting
 - Isometric quadriceps femoris muscle contraction (Powers, Landel and Perry, 1996)
-
- ❖ A symptomatic sacroiliac (SI) joint on the ipsilateral side. If the patient had bilateral PFPS, then the patient was asked which knee was more painful. The diagnosis of a symptomatic SI joint was guided by the following signs: if there was pain over the posterior superior iliac spine (PSIS) and if provocation tests such as Gaenslen's, Patrick's Faber, Erickson's and Yeoman's test, exacerbate discomfort over the PSIS (Kirkaldy-Willis and Burton, 1992:125). SI joint dysfunction also needed to be present (Suter *et al.*, 1999).

3.3.1.2 Exclusion criteria

The following list was compiled from: Gatterman, 1990:142; Powers, Landel and Perry, 1996; Kannus *et al.*, 1999; Rowlands and Brantingham, 1999.

Subjects will be excluded if they have a history of:

- Traumatic or spontaneous patellar dislocation;
- Any neurological involvement that influenced their gait; or
- Had undergone knee surgery.

Subjects presenting with any of the following were also be excluded:

- Bursitis (suprapatellar, prepatellar, subcutaneous infrapatellar, deep infrapatellar bursitis over the tibial tuberosity).
- Fat pad syndrome.
- Any systemic arthritides that affect the knee.
- Evidence of a meniscal tear.
- Ligamentous instability.
- Abnormalities indicative of osteochondritis dessicans, osteoarthritis or loose bodies.
- Pregnant and breast-feeding subjects.
- Suspected disc herniations with increasing signs and symptoms of neurological deficit.
- Suspected abdominal aortic aneurysm.
- Suspected lumbar spine tumours.
- Suspected lumbar spine infections.
- Traumatic lumbar spine injuries.

Patients presenting with acute, severe PFPS experiencing pain that would prevented them from completing the isokinetic testing were also excluded from the study (Cybex, 1996: 1-13).

3.3.2 Allocation of subjects

Thirty subjects were accepted into the study and allocated into three groups of ten subjects each, using a convenience, purposive sampling approach. Subjects in group A received a long axis distraction manipulation of the tibio-femoral joint; subjects in group B received a sacroiliac joint manipulation on the ipsilateral side of the knee pain; and subjects in group C received both a long axis distraction manipulation of the tibio-femoral joint and a sacroiliac joint manipulation on the ipsilateral side of the knee pain. Subjects who suffered from bilateral PFPS were asked to make a subjective judgement as to which knee was worse, only that knee was then tested.

3.3.3 The first consultation

After the initial telephone discussion, an appointment was made at the Durban Institute of Technology Chiropractic Clinic. Subjects then underwent a full case history, physical, lumbar spine and knee regional examinations (see Appendix B). Subjects accepted into the study were given a covering letter (see Appendix C), explaining the study to them. They were also required to complete and sign an informed consent form (see Appendix D). Subjects had to be diagnosed with PFPS. Subjects in groups B and C also had to present with a symptomatic sacroiliac joint. Subjects were asked to fill out a Numerical Pain Rating Scale (NPRS).

3.3.4 The second consultation

Subjects were screened for any change in their condition, which would prevent them from undergoing isokinetic testing or spinal/extra-spinal manipulative therapy, since the initial visit. If a change in their condition, with respect to the symptomatic SI and knee joints, was noted, they were excluded from the study.

Subjects completed a 5 minute warm-up cycle, followed by 3 sets of a 20 second hamstring and quadriceps muscle stretch. The subject then underwent concentric-concentric testing of the thigh.

Once all baseline measurements were taken, subjects in group A received a long axis distraction manipulation of the tibio-femoral joint; subjects in group B received a sacroiliac joint manipulation on the ipsilateral side of the knee pain; and subjects in group C received both a long axis distraction manipulation of the tibio-femoral joint and an sacroiliac joint manipulation on the ipsilateral side of the knee pain. For the subjects in group C, the tibio-femoral joint was always manipulated, first followed by the sacroiliac joint manipulation.

Following this, another NPRS was filled out and the concentric-concentric contraction of the thigh was re-tested. This was done in order to allow for comparison of the muscle strength and NPRS values pre- and post-manipulation.

Suter *et al.* (2000) found that changes in knee-extensor strength in the contralateral legs of both the treatment and control groups, following SI joint manipulation, did not reach a level of statistical significance. Therefore, only the ipsilateral thigh underwent concentric-concentric muscle testing.

3.4 METHODS OF MEASUREMENT

3.4.1 The data

3.4.1.1 Primary data

A measurement of the subjects quadriceps muscle strength was obtained pre and post manipulation, using a Cybex Orthotron II Isokinetic Rehabilitation System. The subjective awareness of the subject's level of pain (related to the PFPS) was also measured pre and post manipulation, using the Numerical Pain Rating Scale (NPRS) (Jensen, Karoly and Braver, 1986).

3.4.1.2 Secondary data

The secondary data was obtained from a search of the related literature. This included journals, articles, published reports, web sites and textbooks, containing information relevant to the research being conducted.

3.4.2 Objective measurement: concentric-concentric isokinetic testing

The Cybex Orthotron II Isokinetic Rehabilitation System was used to perform the concentric-concentric isokinetic testing of the thigh. This machine is designed to measure the voluntary force output (isometric contractibility) of a muscle group. The muscles tested were the quadriceps and hamstrings. Several authors have agreed on the reliability and validity of this instrument (Davies, 1992: 35; Chan and Mafulli, 1996: 22-3; Callaghan *et al.*, 2000).

Before the isokinetic testing was performed, subjects completed a 5 minute warm-up cycle, followed by 3 sets of a 20 second hamstring and quadriceps stretch. Subjects were placed in a comfortable upright-seated position, back rest at 85⁰, and secured using thigh, pelvic and torso straps in order to minimize

extraneous body movements. The knee rested at an angle of 90° from full extension. The lateral femoral condyle was used as the bony landmark for matching the axis of rotation of the knee joint with the axis of rotation of the dynamometer resistance adaptor. Subjects were given verbal encouragement while performing the test. The concentric-concentric testing procedure was as follows:

- 6 sub-maximal warm-up repetitions at $90^{\circ}/\text{sec}$
- 1 minute rest
- 3-5 repetitions of maximal effort at $60^{\circ}/\text{sec}$
- 5 minute warm-down cycle
- Manipulative intervention/s applied
- 3-5 repetitions of maximal effort at $60^{\circ}/\text{sec}$

[Davies, 1992:43-4; Perrin, 1993:48; Chan and Muffini, 1996:10; Pincivero, Lephart and Karunakara, 1997; Suter *et al.*, 2000; Clifton, 2003]

An average of three readings -before and after manipulation- were taken in order to make an accurate measurement of the isometric contractibility of the thigh (Ringdahl, 1993:132).

3.4.3 Subjective measurement: numerical pain rating scale (NPRS)

This scale is used to ascertain the pain intensity that the subjects experienced. A numerical scale from zero to one hundred was used, where zero indicated “No pain at all” and one hundred indicated, “Pain as bad as it could be”. The questionnaire instructed the subjects to rate their pain when it was at its worst and at it’s least. An accurate assessment of the pain intensity was then obtained by taking the average of the two scores (Jensen, Karoly and Braver, 1986).

An average of the amount of pain experienced by each subject was taken pre and post manipulation. Jensen, Karoly and Braver (1986) state that the NPRS is a reliable method of rating pain intensity.

3.5 INTERVENTIONS

3.5.1 Manipulative therapy

Three manipulative treatment protocols were tested during this study. Subjects in group A received a long axis distraction manipulation of the tibio-femoral joint; subjects in group B received a sacroiliac (SI) joint manipulation on the ipsilateral side of the knee pain; and subjects in group C received both a long axis distraction manipulation of the tibio-femoral joint and a joint manipulation on the ipsilateral side of the knee pain.

Only the symptomatic knee was manipulated. If a subject complained of bilateral PFPS, they were asked to make a subjective judgement as to which knee was worse (Suter *et al.*, 2000). That knee was then manipulated. An audible cavitation was not required to indicate a successful manipulation (Suter *et al.*, 1994).

Restrictions in the knee joint were identified by using dynamic palpation of the knee, as described by Schafer and Faye (1990: 393-398). A long-axis distraction manipulation was used to correct the fixation and to stimulate mechanoreceptors around the knee joint (Schafer and Faye, 1990: 40).

The SI joint was manipulated according to the restriction palpated. Restrictions in the SI joint occur in either the upper or lower area of the joint. These areas may be restricted in flexion or extension. The standing flexed-knee-raising test was used to identify the restriction present in the SI joint (Schafer and Faye, 1990: 260).

The manipulation techniques employed were all diversified manipulations according to Szaraz (1990: 139-141) and Schafer and Faye (1990: 393), which are outlined below.

3.5.1.1 The long-axis distraction manipulation of the tibio-femoral joint

The patient was required to lie supine. The researcher contacted his hands bilaterally just distal to the tibio-femoral joint space. The patients' distal leg was secured at approximately the malleoli level between the researchers' knees. The patients' knee was placed into slight flexion, by elevating the researchers' hands, whilst he performed a partial deep knee bend. The manipulation was achieved by allowing the patients' knee to drop into extension and exerting a long-axis distraction through an inferior line of drive, by a sharp snapping of the knees into extension, assisted by a caudal pull through the researchers hand contacts. (Meyer *et al.*, 1990; Schafer and Faye, 1990: 393).

3.5.1.2 The sacroiliac (SI) joint manipulation

The positioning of the patient for the manipulation was as follows:

Flexion fixation – patient in side lying position with the lesion side up.

Extension fixation – patient in side lying position with the lesion side down.

Once the patient was in a side lying position, the patients' lower arm was pulled towards the researcher, until the patient was in a comfortable position. The lower arm was then folded over the top of the shoulder and stabilized with the researchers' cephalad hand. The patients' lower leg was slightly bent at the knee and the upper leg flexed at the hip and knee. The foot of the upper leg was placed into the popliteal space of the lower leg, ensuring that the pelvis was at 90 degrees to the table.

The researcher stood alongside the patient in a Fencer stance. The patients' bent upper leg was supported between the researchers' upper thighs. The researcher then took a pisiform contact with his caudal hand over the involved area of the SI joint (superior/inferior). The researcher then removed the slack by applying a cephalid traction force with his indifferent hand and an anterior rotation of the pelvis with the contact hand. Once all the slack had been

removed, the researcher then did a body drop and applied a high velocity, low amplitude thrust, in an inferior line of drive.

[Adapted from Szaraz, 1990: 139-141]

3.6 STATISTICAL ANALYSIS

Non-parametric testing was used to analyse the data, as the sample group consisted of thirty subjects.

3.6.1 Intra-group comparisons

Both the subjective and objective data were tested for a significant change as a result of the intervention, using the Wilcoxon Signed Ranks test for matched pairs.

3.6.2 Inter-group comparisons

The Kruskal-Wallis H test was used to test for a significant difference between the treatment groups, for all variables tested.

3.6.3 Correlation comparisons

The Pearson's Product Moment Correlation coefficient was applied to test for a significant relationship between the variables.

The level of significance will be set at $\alpha = 0,05$ and p-values will be used for decision-making regarding the null hypothesis. All data will be analysed using the SPSS package.

The objective data was treated as follows:

- The Cybex readings were recorded separately for each group.
- The quadriceps and hamstring ratios were then calculated using the average of three maximum voluntary force readings¹.
- The data was analysed using a 95% confidence level.

¹ The quadriceps ratio = (quadriceps force/ quadriceps + hamstring force)*100
The hamstring ratio = (hamstring force/quadriceps +hamstring force)*100

The subjective data was treated as follows:

- Questionnaires that the patients completed were screened to ensure that they had been completed correctly.
- Raw data from the questionnaires were recorded separately for each group.
- The data was analysed using a 95% confidence level.

3.7 STATISTICAL PROCEDURE

The statistical package SPSS was used for capture and analysis. The following tests were used:

- The Wilcoxon Signed Ranks Test for matched pairs within Groups A, B and C
- The Kruskal-Wallis H Test between Groups A, B and C.
- The Pearson Product Moment Correlation Coefficient was used to test for a significant relationship between variables.

3.7.1 Intra-group comparisons: Non-parametric paired testing

The Wilcoxon Signed Ranks test for matched pairs was used to compare both the objective and subjective results from related samples. In each test, the null hypothesis states that there is no significant improvement between the two related samples being compared at the α level of significance. The alternative hypothesis states that there is a significant improvement.

According to Zar (1996: 662), the Wilcoxon Signed Ranks test has its most significant application in paired sample testing.

Decision rule:

The null hypothesis is rejected at the $\alpha = 0.05$ level of significance if $p \leq \alpha$, where p is the observed level or probability value. Otherwise, the null hypothesis is accepted at the same level.

3.7.2 Inter-group comparisons: Non-Parametric Paired Tests

The Kruskal-Wallis H test was used to compare Groups A, B and C with respect to each categorical variable (both objective and subjective). The null hypothesis states that there is no significant difference between Groups A, B and C with respect to the variable of comparison at the $\alpha = 0.05$ level of significance. The alternative hypothesis states that there is a significance difference at the same level of significance.

Decision rule:

The null hypothesis is rejected at the α level of significance if $p \leq \alpha$, where p is the observed level of probability value. Otherwise, the null hypothesis is accepted at the same level.

3.7.3 Correlation comparisons

The Pearson Product Moment Correlation coefficient was applied to test for a significant relationship between the variables. The null hypothesis states that there is no significant relationship between the different variables at the $\alpha = 0.05$ level of significance. The alternative hypothesis states that there is a significant relationship at the same level of significance.

Decision rule:

The null hypothesis is rejected at the α level of significance if $p \leq \alpha$, where p is the observed level of probability value. Otherwise, the null hypothesis is accepted at the same level.

CHAPTER FOUR

RESULTS

4.1 INTRODUCTION

This chapter summarizes the results obtained from the primary data collected over the duration of the study. Measurements included:

- Maximum voluntary quadriceps force output -measured in Newton meters (NM).
- Maximum voluntary hamstring force output -measured in Newton meters (NM).
- The quadriceps ratio -expressed as a percentage
- The hamstring ratio -expressed as a percentage.
- The level of perceived pain, using the numerical pain rating scale (NPRS).

4.2 SAMPLE SIZE

The sample size consisted of 30 patients that were allocated into three treatment groups. Convenience purposive sampling was used to allocate the subjects into the treatment groups. Subjects in group A received a long axis distraction manipulation of the tibio-femoral joint, subjects in group B received a sacroiliac joint manipulation on the ipsilateral side of the knee pain, and subjects in group C received both a long axis distraction manipulation of the tibio-femoral joint and a sacroiliac joint manipulation on the ipsilateral side of the knee pain.

4.3 DEMOGRAPHICS

The following tables summarize the age, race and gender distribution of the sample and the individual treatment groups.

4.3.1 Age distribution

Table 4.1 Age distributions.

| <u>Age distribution of sample</u> | | | |
|--|-----------|------------------|------------|
| Age group distribution | Frequency | Mean age (years) | Percentage |
| 18-25 | 15 | 22.87 | 50 |
| 26-35 | 13 | 29.38 | 43.33 |
| 36-40 | 2 | 39.00 | 6.67 |

| <u>Age distribution within the treatment groups</u> | | |
|--|---------|------|
| Group A | N | 10 |
| | Mean | 26.5 |
| | Minimum | 21 |
| | Maximum | 39 |
| Group B | N | 10 |
| | Mean | 28.9 |
| | Minimum | 23 |
| | Maximum | 39 |
| Group C | N | 10 |
| | Mean | 24.9 |
| | Minimum | 20 |
| | Maximum | 34 |

Table 4.2 Age distribution of the sample group.

| <u>Age distribution of the sample</u> | |
|--|-------|
| | Years |
| Number of subjects | 30 |
| Mean age | 26.77 |
| Minimum age | 20 |
| Maximum age | 39 |

4.3.2 Race and gender distributions

Table 4.3 Race distributions.

| <u>Race distribution</u> | | | |
|---|--------|-----------|---------|
| Race distribution of the treatment groups | | | |
| Group | | Frequency | Percent |
| Group A | White | 6 | 60 |
| | Black | 2 | 20 |
| | Indian | 2 | 20 |
| | Total | 10 | 100 |
| Group B | White | 7 | 70 |
| | Black | 1 | 10 |
| | Indian | 2 | 20 |
| Total | | 10 | 100 |
| Group C | White | 10 | 100 |

| Race distribution of the sample | | |
|---------------------------------|-----------|---------|
| | Frequency | Percent |
| White | 23 | 76.67 |
| Black | 3 | 10.00 |
| Indian | 4 | 13.33 |
| Total | 30 | 100.00 |

Table 4.4 Gender distributions.

| <u>Gender distribution</u> | | | |
|---|--------|-----------|---------|
| Gender distribution of the treatment groups | | | |
| Group | | Frequency | Percent |
| Group A | Male | 7 | 70 |
| | Female | 3 | 30 |
| | Total | 10 | 100 |
| Group B | Male | 5 | 50 |
| | Female | 5 | 50 |
| | Total | 10 | 100 |
| Group C | Male | 4 | 40 |
| | Female | 6 | 60 |
| | Total | 10 | 100 |

| Gender distribution of the sample | | |
|-----------------------------------|-----------|---------|
| | Frequency | Percent |
| Male | 16 | 53.33 |
| Female | 14 | 46.67 |
| Total | 30 | 100.00 |

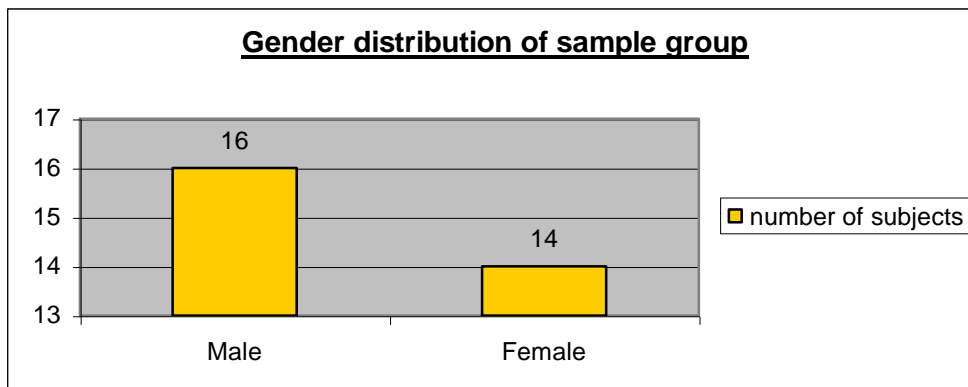


Figure 4.1 Gender distribution of the sample group.

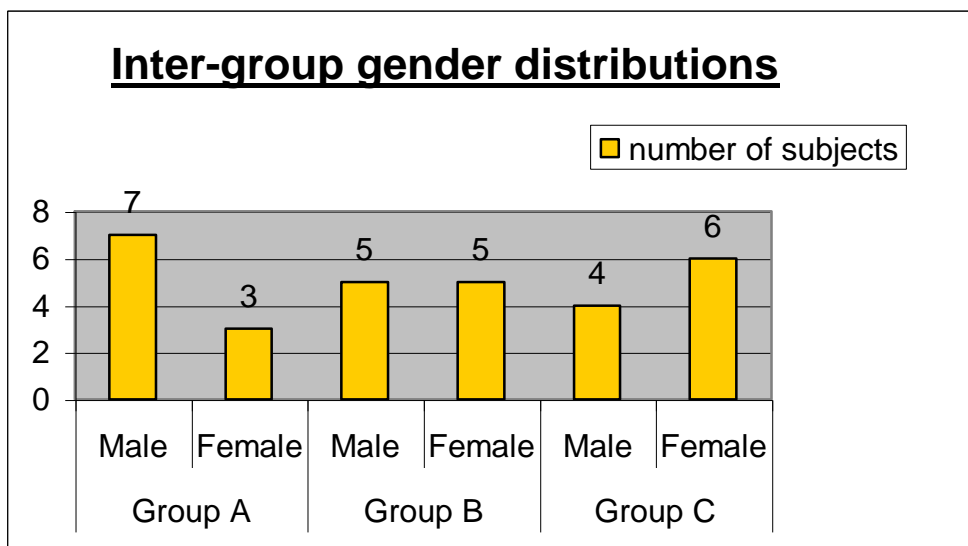


Figure 4.2 Inter-group gender distributions.

4.3.3 Activity comparisons

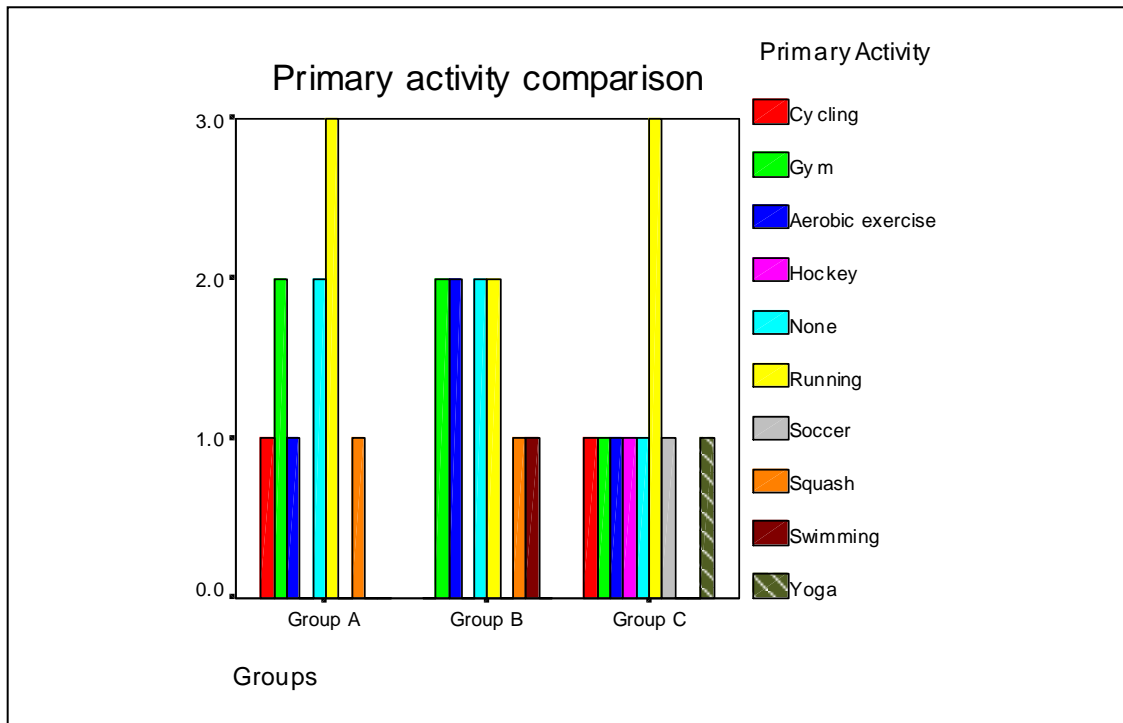


Figure 4.3 Primary activity comparisons.

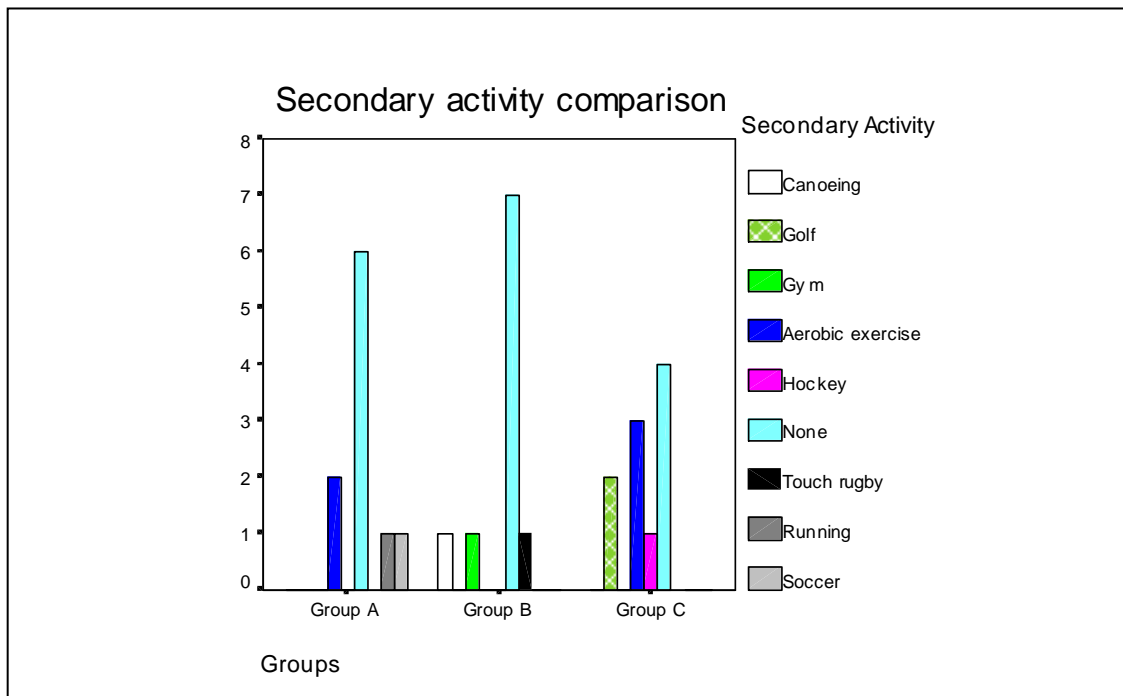


Figure 4.4 Secondary activity comparisons.

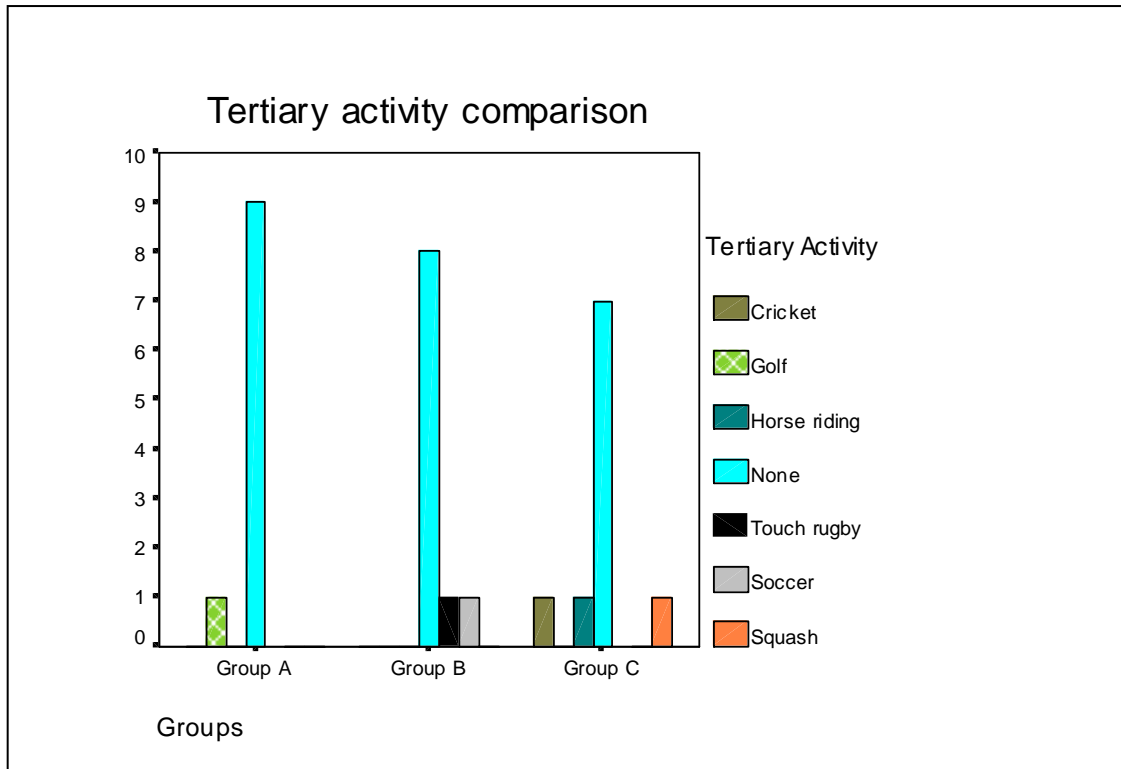


Figure 4.5 Tertiary activity comparisons.

Table 4.5 Activity comparisons of the sample.

| | Primary | Secondary | Tertiary |
|------------------|---------|-----------|----------|
| Aerobic gym work | 13 | 17 | 0 |
| Canoeing | 0 | 3 | 0 |
| Cricket | 0 | 0 | 3 |
| Cycling | 7 | 0 | 0 |
| Golf | | 7 | 3 |
| Gym | 17 | 3 | 0 |
| Hockey | 3 | 3 | 0 |
| Horse riding | 0 | 0 | 3 |
| None | 17 | 57 | 80 |
| Running | 27 | 3 | 0 |
| Soccer | 3 | 3 | 3 |
| Squash | 7 | 0 | 3 |
| Swimming | 3 | 0 | 0 |
| Touch rugby | 0 | 3 | 3 |
| Yoga | 3 | 0 | 0 |

4.4 ANALYSIS OF DATA

4.4.1 Intra-group analysis

Non-parametric tests were applied as the sample group consisted of thirty subjects. The Wilcoxon Signed Ranks test for matched pairs was used to compare results between the treatment groups.

4.4.1.1 Maximum voluntary quadriceps force output (NM)

Table 4.6 Descriptive statistics of the quadriceps force.

| Average quadriceps force (NM): Descriptive statistics | | | | | |
|---|--------------------|-------|----------------|-----|-----|
| Group | | Mean | Std. Deviation | Min | Max |
| Group A | Pre -Manipulation | 186.2 | 60.76 | 77 | 302 |
| | Post- Manipulation | 187.5 | 50.68 | 91 | 266 |
| Group B | Pre -Manipulation | 129.8 | 36.15 | 87 | 186 |
| | Post- Manipulation | 143.7 | 54.76 | 75 | 232 |
| Group C | Pre -Manipulation | 159.3 | 59.31 | 102 | 274 |
| | Post- Manipulation | 167.8 | 63.13 | 108 | 302 |

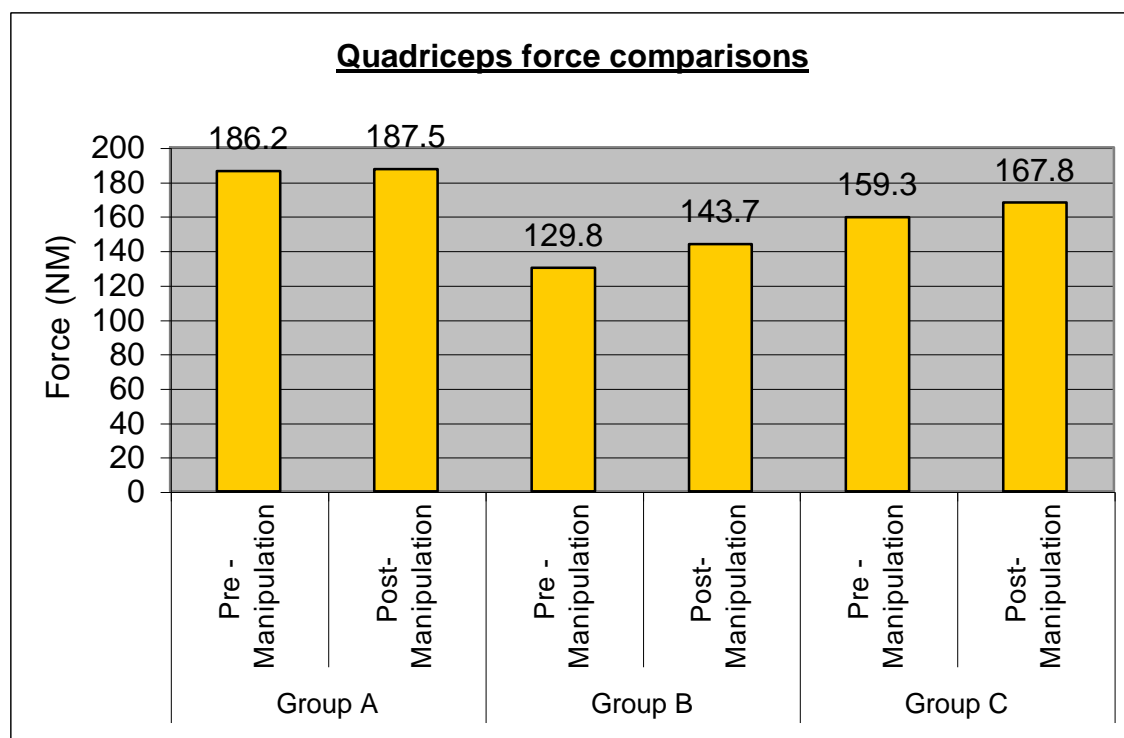


Figure 4.6 Quadriceps force comparisons.

Table 4.7 Wilcoxon Signed Ranks test for matched pairs: quadriceps force.

| Average quadriceps force : Wilcoxon Signed Ranks Test | | | | | |
|---|-----------------------------|----|-----------|--------------|--------------|
| Group | | N | Mean Rank | Sum of Ranks | P-Value |
| Group A | Negative Ranks ^a | 3 | 6.50 | 19.50 | 0.415 |
| | Positive Ranks ^b | 7 | 5.07 | 35.50 | |
| | Ties ^c | 0 | | | |
| | Total | 10 | | | |
| Group B | Negative Ranks ^a | 1 | 6.00 | 6.00 | 0.050 |
| | Positive Ranks ^b | 8 | 4.88 | 39.00 | |
| | Ties ^c | 1 | | | |
| | Total | 10 | | | |
| Group C | Negative Ranks ^a | 3 | 5.50 | 16.50 | 0.262 |
| | Positive Ranks ^b | 7 | 5.50 | 38.50 | |
| | Ties ^c | 0 | | | |
| | Total | 10 | | | |

a Average quadriceps force (Post- Manipulation) < Average quadriceps force (Pre -Manipulation)
b Average quadriceps force (Post- Manipulation) > Average quadriceps force (Pre -Manipulation)
c Average quadriceps force (Pre -Manipulation) = Average quadriceps force (Post- Manipulation)

For Group B, the null hypothesis was rejected because $p \leq 0.05$. This indicates a significant improvement in the quadriceps force after the sacroiliac manipulation on the ipsilateral side of the knee pain.

For Groups A and C, the null hypothesis was accepted because $p > 0.05$. Therefore, there was no significant improvement in the quadriceps force after manipulation of the knee joint and a combination of manipulations of the knee and sacroiliac joints.

4.4.1.2 Maximum voluntary hamstring force output

Table 4.8 Descriptive statistics of the hamstring force.

| Average hamstring force (NM): Descriptive statistics | | | | | |
|--|--------------------|------|----------------|-----|-----|
| Group | | Mean | Std. Deviation | Min | Max |
| Group A | Pre- Manipulation | 107 | 26.85 | 71 | 156 |
| | Post- Manipulation | 114 | 31.59 | 64 | 163 |
| Group B | Pre- Manipulation | 81.4 | 26.07 | 58 | 133 |
| | Post- Manipulation | 89.4 | 33.49 | 62 | 146 |
| Group C | Pre- Manipulation | 97.6 | 31.94 | 62 | 163 |
| | Post- Manipulation | 105 | 35.22 | 68 | 172 |

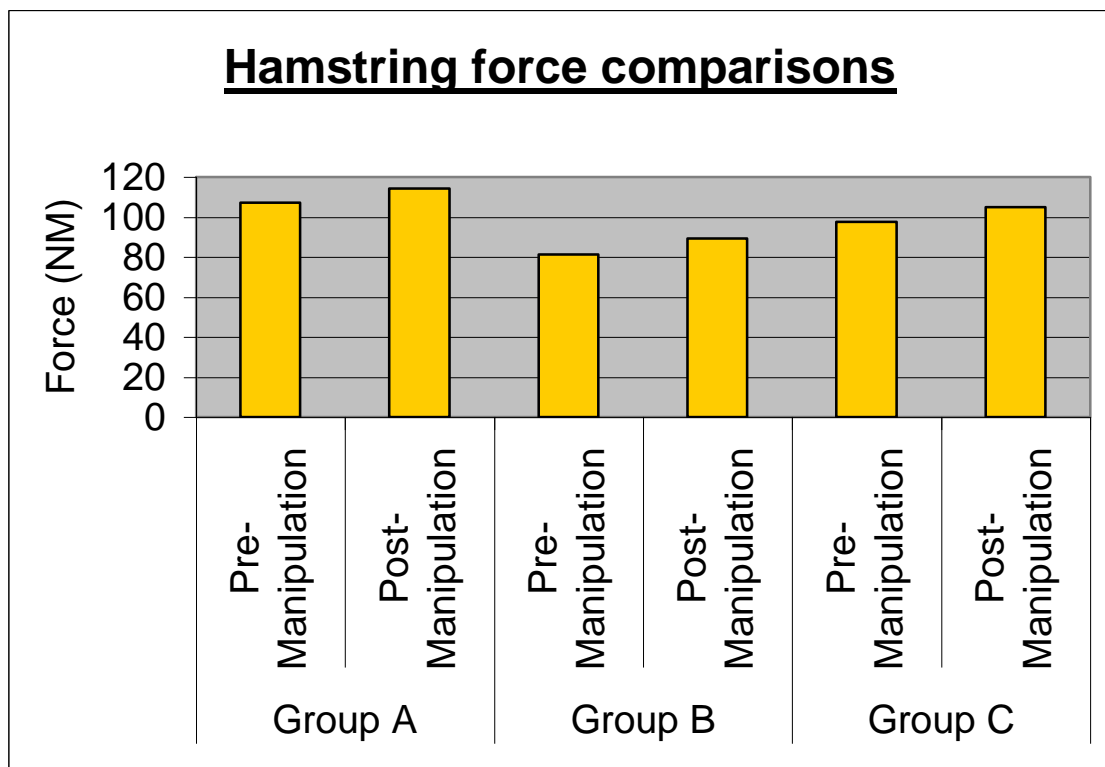


Figure 4.7 Hamstring force comparisons.

Table 4.9 Wilcoxon Signed Ranks test for matched pairs: hamstring force.

| Average hamstrings force : Wilcoxon Signed Ranks Test | | | | | |
|---|-----------------------|----|-----------|--------------|--------------|
| Group | Rank | N | Mean Rank | Sum of Ranks | P-Value |
| Group A | Negative ^a | 2 | 3.50 | 7 | 0.123 |
| | Positive ^b | 6 | 4.83 | 29 | |
| | Ties ^c | 2 | | | |
| | Total | 10 | | | |
| Group B | Negative ^a | 2 | 3.50 | 7 | 0.036 |
| | Positive ^b | 8 | 6.00 | 48 | |
| | Ties ^c | 0 | | | |
| | Total | 10 | | | |
| Group C | Negative ^a | 2 | 1.50 | 3 | 0.021 |
| | Positive ^b | 7 | 6.00 | 42 | |
| | Ties ^c | 1 | | | |
| | Total | 10 | | | |

a Average Hamstring force (Post- Manipulation) < Average Hamstring force (Pre- Manipulation)
b Average Hamstring force (Post- Manipulation) > Average Hamstring force (Pre- Manipulation)
c Average Hamstring force (Pre- Manipulation) = Average Hamstring force (Post- Manipulation)

For Groups B and C, the null hypothesis was rejected because $p \leq 0.05$. This indicates a significant improvement in the hamstring force after the sacroiliac manipulation on the ipsilateral side of the knee pain and a combination of manipulations of the knee and sacroiliac joints.

For Group A, the null hypothesis was accepted because $p > 0.05$. Therefore, there was no significant improvement in the quadriceps force after manipulation on the knee joint.

4.4.1.3 The quadriceps ratio

Table 4.10 Descriptive statistics of the quadriceps ratios.

| Quadriceps ratio (%): Descriptive statistics | | | | | |
|--|--------------------|-------|----------------|-------|-------|
| Group | | Mean | Std. Deviation | Min | Max |
| Group A | Pre- Manipulation | 62.73 | 4.99 | 52.03 | 70.07 |
| | Post- Manipulation | 62.07 | 2.81 | 57.96 | 67.80 |
| Group B | Pre- Manipulation | 61.58 | 3.66 | 54.38 | 66.15 |
| | Post- Manipulation | 61.39 | 3.42 | 54.74 | 66.83 |
| Group C | Pre- Manipulation | 61.71 | 4.20 | 52.51 | 67.82 |
| | Post- Manipulation | 61.31 | 3.88 | 52.86 | 66.96 |

Table 4.11 Wilcoxon Signed Ranks test for matched pairs: quadriceps ratios.

| Average quadriceps ratio : Wilcoxon Signed Ranks Test | | | | | |
|---|-----------------------|----|-----------|--------------|---------|
| Group | Rank | N | Mean Rank | Sum of Ranks | P-Value |
| Group A | Negative ^a | 7 | 5.57 | 39 | 0.241 |
| | Positive ^b | 3 | 5.33 | 16 | |
| | Ties ^c | 0 | | | |
| | Total | 10 | | | |
| Group B | Negative ^a | 7 | 4.43 | 31 | 0.721 |
| | Positive ^b | 3 | 8.00 | 24 | |
| | Ties ^c | 0 | | | |
| | Total | 10 | | | |
| Group C | Negative ^a | 6 | 6.50 | 39 | 0.241 |
| | Positive ^b | 4 | 4.00 | 16 | |
| | Ties ^c | 0 | | | |
| | Total | 10 | | | |

a Quadriceps ratio (Post- Manipulation) < Quadriceps ratio (Pre- Manipulation)
b Quadriceps ratio (Post- Manipulation) > Quadriceps ratio (Pre- Manipulation)
c Quadriceps ratio (Pre- Manipulation) = Quadriceps ratio (Post- Manipulation)

For all groups, the null hypothesis was accepted as $p \leq 0.05$. This indicates that there was no significant change in the quadriceps ratio after any of the three different manipulative treatment protocols.

4.4.1.4 The hamstring ratio

Table 4.12 Descriptive statistics of the hamstring ratios.

| Group | | Mean | Std. Deviation | Min | Max |
|---------|--------------------|-------|----------------|-------|-------|
| Group A | Pre- Manipulation | 37.27 | 4.99 | 29.93 | 47.97 |
| | Post- Manipulation | 37.93 | 2.81 | 32.20 | 42.04 |
| Group B | Pre- Manipulation | 38.42 | 3.66 | 33.85 | 45.63 |
| | Post- Manipulation | 38.61 | 3.42 | 33.17 | 45.26 |
| Group C | Pre- Manipulation | 38.29 | 4.20 | 32.18 | 47.49 |
| | Post- Manipulation | 38.69 | 3.88 | 33.04 | 47.14 |

Table 4.13 Wilcoxon Signed Ranks test for matched pairs: hamstring ratios.

| Group | Rank | N | Mean Rank | Sum of Ranks | P-Value |
|---------|-----------------------|----|-----------|--------------|---------|
| Group A | Negative ^a | 3 | 5.33 | 16 | 0.241 |
| | Positive ^b | 7 | 5.57 | 39 | |
| | Ties ^c | 0 | | | |
| | Total | 10 | | | |
| Group B | Negative ^a | 3 | 8 | 24 | 0.721 |
| | Positive ^b | 7 | 4.43 | 31 | |
| | Ties ^c | 0 | | | |
| | Total | 10 | | | |
| Group C | Negative ^a | 4 | 4 | 16 | 0.241 |
| | Positive ^b | 6 | 6.50 | 39 | |
| | Ties ^c | 0 | | | |
| | Total | 10 | | | |

a Hamstring ratio (Post- Manipulation) < Hamstring ratio (Pre- Manipulation)
b Hamstring ratio (Post- Manipulation) > Hamstring ratio (Pre- Manipulation)
c Hamstring ratio (Pre- Manipulation) = Hamstring ratio (Post- Manipulation)

For all groups, the null hypothesis was accepted as $p \leq 0.05$. This indicates that there was no significant change in the hamstring ratio after any of the three different manipulative treatment protocols.

4.4.1.5 Level of perceived pain using the numerical pain rating scale (NPRS)

Table 4.14 Descriptive statistics of the NPRS.

| Group | | Mean | Std. Deviation | Min | Max |
|---------|--------------------|-------|----------------|-----|------|
| Group A | Pre- Manipulation | 39.85 | 5.86 | 30 | 47.5 |
| | Post- Manipulation | 31.75 | 17.04 | 7.5 | 65 |
| Group B | Pre- Manipulation | 37.5 | 15.50 | 10 | 60 |
| | Post- Manipulation | 33.75 | 18.23 | 10 | 60 |
| Group C | Pre- Manipulation | 40.75 | 10.07 | 25 | 55 |
| | Post- Manipulation | 37 | 15.63 | 15 | 60 |

Table 4.15 Wilcoxon Signed Ranks test for matched pairs: NPRS.

| Group | Rank | N | Mean Rank | Sum of Ranks | P-Value |
|---------|-----------------------|----|-----------|--------------|---------|
| Group A | Negative ^a | 6 | 5.42 | 32.5 | 0.236 |
| | Positive ^b | 3 | 4.17 | 12.5 | |
| | Ties ^c | 1 | | | |
| | Total | 10 | | | |
| Group B | Negative ^a | 5 | 4.60 | 23 | 0.121 |
| | Positive ^b | 2 | 2.5 | 5 | |
| | Ties ^c | 3 | | | |
| | Total | 10 | | | |
| Group C | Negative ^a | 4 | 5.13 | 20.5 | 0.271 |
| | Positive ^b | 3 | 2.5 | 7.5 | |
| | Ties ^c | 3 | | | |
| | Total | 10 | | | |

a Average NPRS (Post- Manipulation) < Average NPRS reading (Pre- Manipulation)
b Average NPRS (Post- Manipulation) > Average NPRS reading (Pre- Manipulation)
c Average NPRS reading (Pre- Manipulation) = Average NPRS (Post- Manipulation)

For all groups, the null hypothesis was accepted as $p \leq 0.05$. This indicates that there was no significant change in NPRS after any of the three different manipulative treatment protocols.

4.4.2 Inter-group analysis

Non-parametric tests were applied as the sample consisted of thirty subjects. The Kruskal-Wallis H test was used to test for a significant difference between the treatment groups.

4.4.2.1 Pre manipulation

Table 4.16 Kruskal-Wallis H test: pre manipulation.

| Inter-group comparison: Pre-manipulation | | | | |
|--|---------|----|-----------|----------|
| Variable | Group | N | Mean Rank | P- value |
| Average quadriceps force | Group A | 10 | 20.10 | 0.083 |
| | Group B | 10 | 11.35 | |
| | Group C | 10 | 15.05 | |
| | Total | 30 | | |
| Average hamstring force | Group A | 10 | 19.55 | 0.087 |
| | Group B | 10 | 10.90 | |
| | Group C | 10 | 16.05 | |
| | Total | 30 | | |
| quadriceps ratio | Group A | 10 | 17.40 | 0.704 |
| | Group B | 10 | 14.65 | |
| | Group C | 10 | 14.45 | |
| | Total | 30 | | |
| hamstring ratio | Group A | 10 | 13.60 | 0.704 |
| | Group B | 10 | 16.35 | |
| | Group C | 10 | 16.55 | |
| | Total | 30 | | |
| Numerical pain rating scale | Group A | 10 | 15.75 | 0.878 |
| | Group B | 10 | 14.40 | |
| | Group C | 10 | 16.35 | |
| | Total | 30 | | |

The null hypothesis was accepted, as none of the P-values were ≤ 0.05 . This indicates that there was no statistical difference pre-manipulation, between the treatment groups for any of the variables tested.

4.4.2.2 Post manipulation

Table 4.17 Kruskal-Wallis H test: post manipulation.

| Inter-group comparison: Post-manipulation | | | | |
|---|---------|----|-----------|----------|
| Variable | Group | N | Mean Rank | P- value |
| Average quadriceps force | Group A | 10 | 19.30 | 0.174 |
| | Group B | 10 | 11.95 | |
| | Group C | 10 | 15.25 | |
| | Total | 30 | | |
| Average hamstring force | Group A | 10 | 18.65 | 0.173 |
| | Group B | 10 | 11.45 | |
| | Group C | 10 | 16.40 | |
| | Total | 30 | | |
| quadriceps ratio | Group A | 10 | 16.90 | 0.818 |
| | Group B | 10 | 15.10 | |
| | Group C | 10 | 14.50 | |
| | Total | 30 | | |
| hamstring ratio | Group A | 10 | 14.10 | 0.818 |
| | Group B | 10 | 15.90 | |
| | Group C | 10 | 16.50 | |
| | Total | 30 | | |
| Numerical pain rating scale | Group A | 10 | 13.85 | 0.729 |
| | Group B | 10 | 15.70 | |
| | Group C | 10 | 16.95 | |
| | Total | 30 | | |

The null hypothesis was accepted, as none of the P-values were ≤ 0.05 . This indicates that there was no statistical difference post-manipulation, between the groups for any of the variables tested.

4.4.3 Correlation comparisons

The Pearson Product Moment Correlation coefficient was applied to test for any correlation between the variables.

4.4.3.1 Pre manipulation correlations

Table 4.18 Pre manipulation correlations.

| | | | Pre-manipulation correlations | | | | |
|----------------------|--------------------------|---------------------|-------------------------------|-------------------------|------------------|-----------------|----------------------|
| Group | | | Average quadriceps force | Average hamstring force | Quadriceps ratio | Hamstring ratio | Average NPRS reading |
| Group A | Average quadriceps force | Pearson Correlation | 1.000 | .778** | .712* | -.712* | .234 |
| | | Sig. (2-tailed) | . | .008 | .021 | .021 | .514 |
| | | N | 10 | 10 | 10 | 10 | 10 |
| | Average hamstring force | Pearson Correlation | .778** | 1.000 | .182 | -.182 | .063 |
| | | Sig. (2-tailed) | .008 | . | .614 | .614 | .863 |
| | | N | 10 | 10 | 10 | 10 | 10 |
| | Quadriceps ratio | Pearson Correlation | .712* | .182 | 1.000 | -1.000** | -.016 |
| | | Sig. (2-tailed) | .021 | .614 | . | .000 | .965 |
| | | N | 10 | 10 | 10 | 10 | 10 |
| | Hamstring ratio | Pearson Correlation | -.712* | -.182 | -1.000** | 1.000 | .016 |
| | | Sig. (2-tailed) | .021 | .614 | .000 | . | .965 |
| | | N | 10 | 10 | 10 | 10 | 10 |
| Average NPRS reading | Pearson Correlation | .234 | .063 | -.016 | .016 | 1.000 | |
| | Sig. (2-tailed) | .514 | .863 | .965 | .965 | . | |
| | N | 10 | 10 | 10 | 10 | 10 | |
| Group B | Average quadriceps force | Pearson Correlation | 1.000 | .871** | .079 | -.079 | -.398 |
| | | Sig. (2-tailed) | . | .001 | .829 | .829 | .255 |
| | | N | 10 | 10 | 10 | 10 | 10 |
| | Average hamstring force | Pearson Correlation | .871** | 1.000 | -.403 | .403 | -.481 |
| | | Sig. (2-tailed) | .001 | . | .248 | .248 | .159 |
| | | N | 10 | 10 | 10 | 10 | 10 |
| | Quadriceps ratio | Pearson Correlation | .079 | -.403 | 1.000 | -1.000** | .244 |
| | | Sig. (2-tailed) | .829 | .248 | . | .000 | .496 |
| | | N | 10 | 10 | 10 | 10 | 10 |
| | Hamstring ratio | Pearson Correlation | -.079 | .403 | -1.000** | 1.000 | -.244 |
| | | Sig. (2-tailed) | .829 | .248 | .000 | . | .496 |
| | | N | 10 | 10 | 10 | 10 | 10 |
| Average NPRS reading | Pearson Correlation | -.398 | -.481 | .244 | -.244 | 1.000 | |
| | Sig. (2-tailed) | .255 | .159 | .496 | .496 | . | |
| | N | 10 | 10 | 10 | 10 | 10 | |
| Group C | Average quadriceps force | Pearson Correlation | 1.000 | .842** | .411 | -.411 | .072 |
| | | Sig. (2-tailed) | . | .002 | .237 | .237 | .843 |
| | | N | 10 | 10 | 10 | 10 | 10 |
| | Average hamstring force | Pearson Correlation | .842** | 1.000 | -.131 | .131 | .140 |
| | | Sig. (2-tailed) | .002 | . | .718 | .718 | .700 |
| | | N | 10 | 10 | 10 | 10 | 10 |
| | Quadriceps ratio | Pearson Correlation | .411 | -.131 | 1.000 | -1.000** | -.047 |
| | | Sig. (2-tailed) | .237 | .718 | . | .000 | .898 |
| | | N | 10 | 10 | 10 | 10 | 10 |
| | Hamstring ratio | Pearson Correlation | -.411 | .131 | -1.000** | 1.000 | .047 |
| | | Sig. (2-tailed) | .237 | .718 | .000 | . | .898 |
| | | N | 10 | 10 | 10 | 10 | 10 |
| Average NPRS reading | Pearson Correlation | .072 | .140 | -.047 | .047 | 1.000 | |
| | Sig. (2-tailed) | .843 | .700 | .898 | .898 | . | |
| | N | 10 | 10 | 10 | 10 | 10 | |

** . Correlation is significant at the 0.01 level (2-tailed).

* . Correlation is significant at the 0.05 level (2-tailed).

The null hypothesis for a relationship between the average quadriceps force and the average hamstring force was rejected for all groups, as the P-values were ≤ 0.05 . This indicates that there was a statistical positive correlation pre-manipulation, between the average quadriceps force and average hamstring force in all groups.

The null hypothesis for a relationship between the average quadriceps force and the quadriceps and hamstring ratios was rejected for group A, as the P-value was ≤ 0.05 . This indicates that there was a statistical positive correlation pre-manipulation, between the average quadriceps force and quadriceps (positive correlation) and hamstring (negative correlation) ratios.

The null hypothesis for a relationship between the quadriceps and the hamstring ratios was rejected for all groups, as the P-values were ≤ 0.05 . This indicates that there was a statistical inverse linear correlation pre-manipulation, between quadriceps and hamstring ratios.

All other null hypotheses testing for significant relationships were accepted, as the P-values were ≥ 0.05 . This means that there were no other significant correlations between the variables.

4.4.3.2 Post manipulation comparisons

Table 4.19 Post manipulation correlations.

| | | | Post-manipulation correlations | | | | |
|------------------|--------------------------|---------------------|--------------------------------|-------------------------|------------------|-----------------|--------------|
| Group | | | Average quadriceps force | Average hamstring force | Quadriceps ratio | Hamstring ratio | Average NPRS |
| Group A | Average quadriceps force | Pearson Correlation | 1.000 | .886** | .324 | -.324 | .245 |
| | | Sig. (2-tailed) | . | .001 | .361 | .361 | .495 |
| | | N | 10 | 10 | 10 | 10 | 10 |
| | Average hamstring force | Pearson Correlation | .886** | 1.000 | -.142 | .142 | -.105 |
| | | Sig. (2-tailed) | .001 | . | .695 | .695 | .774 |
| | | N | 10 | 10 | 10 | 10 | 10 |
| Quadriceps ratio | Pearson Correlation | .324 | -.142 | 1.000 | -1.000** | .787** | |
| | Sig. (2-tailed) | .361 | .695 | . | .000 | .007 | |
| | N | 10 | 10 | 10 | 10 | 10 | |
| Hamstring ratio | Pearson Correlation | -.324 | .142 | -1.000** | 1.000 | -.787** | |
| | Sig. (2-tailed) | .361 | .695 | .000 | . | .007 | |
| | N | 10 | 10 | 10 | 10 | 10 | |
| Average NPRS | Pearson Correlation | .245 | -.105 | .787** | -.787** | 1.000 | |
| | Sig. (2-tailed) | .495 | .774 | .007 | .007 | . | |
| | N | 10 | 10 | 10 | 10 | 10 | |
| Group B | Average quadriceps force | Pearson Correlation | 1.000 | .958** | .328 | -.328 | -.560 |
| | | Sig. (2-tailed) | . | .000 | .355 | .355 | .092 |
| | | N | 10 | 10 | 10 | 10 | 10 |
| | Average hamstring force | Pearson Correlation | .958** | 1.000 | .049 | -.049 | -.573 |
| | | Sig. (2-tailed) | .000 | . | .893 | .893 | .084 |
| | | N | 10 | 10 | 10 | 10 | 10 |
| Quadriceps ratio | Pearson Correlation | .328 | .049 | 1.000 | -1.000** | -.051 | |
| | Sig. (2-tailed) | .355 | .893 | . | .000 | .888 | |
| | N | 10 | 10 | 10 | 10 | 10 | |
| Hamstring ratio | Pearson Correlation | -.328 | -.049 | -1.000** | 1.000 | .051 | |
| | Sig. (2-tailed) | .355 | .893 | .000 | . | .888 | |
| | N | 10 | 10 | 10 | 10 | 10 | |
| Average NPRS | Pearson Correlation | -.560 | -.573 | -.051 | .051 | 1.000 | |
| | Sig. (2-tailed) | .092 | .084 | .888 | .888 | . | |
| | N | 10 | 10 | 10 | 10 | 10 | |
| Group C | Average quadriceps force | Pearson Correlation | 1.000 | .859** | .359 | -.359 | .384 |
| | | Sig. (2-tailed) | . | .001 | .308 | .308 | .273 |
| | | N | 10 | 10 | 10 | 10 | 10 |
| | Average hamstring force | Pearson Correlation | .859** | 1.000 | -.155 | .155 | .417 |
| | | Sig. (2-tailed) | .001 | . | .669 | .669 | .231 |
| | | N | 10 | 10 | 10 | 10 | 10 |
| Quadriceps ratio | Pearson Correlation | .359 | -.155 | 1.000 | -1.000** | .087 | |
| | Sig. (2-tailed) | .308 | .669 | . | .000 | .810 | |
| | N | 10 | 10 | 10 | 10 | 10 | |
| Hamstring ratio | Pearson Correlation | -.359 | .155 | -1.000** | 1.000 | -.087 | |
| | Sig. (2-tailed) | .308 | .669 | .000 | . | .810 | |
| | N | 10 | 10 | 10 | 10 | 10 | |
| Average NPRS | Pearson Correlation | .384 | .417 | .087 | -.087 | 1.000 | |
| | Sig. (2-tailed) | .273 | .231 | .810 | .810 | . | |
| | N | 10 | 10 | 10 | 10 | 10 | |

** . Correlation is significant at the 0.01 level (2-tailed).

The null hypothesis for a relationship between the average quadriceps force and the average hamstring force was rejected for all groups, as the P-values were ≤ 0.05 . This indicates that there was a statistical positive correlation post-manipulation, between the average quadriceps force and average hamstring force in all groups.

The null hypothesis for a relationship between the quadriceps and the hamstring ratios was rejected for all groups, as the P-values were ≤ 0.05 . This indicates that there was a statistical positive linear correlation post-manipulation, between quadriceps and hamstring ratios.

The null hypothesis for a relationship between the average numerical pain rating scale (NPRS) score and the quadriceps and the hamstring ratios was rejected for group A, as the P-value was ≤ 0.05 . This indicates that there was a statistical correlation post-manipulation, between the NPRS and the quadriceps (positive linear correlation) and hamstring (negative linear correlation) ratios.

All other null hypotheses testing for significant relationships were accepted, as the P-values were ≥ 0.05 . This means that there were no other significant correlations between the variables post manipulation.

CHAPTER FIVE

DISCUSSION OF THE RESULTS

5.1 INTRODUCTION

This chapter involves the discussion of the demographic data and the results obtained from the statistical analysis of the primary data (the maximum voluntary quadriceps and hamstring force output, the quadriceps and hamstring ratios and the level of perceived pain). Each variable is discussed independently in terms of objective or subjective outcomes and intra- and inter-group analysis. Any significant correlations between variables will be discussed at the end of this chapter.

5.2 THE DEMOGRAPHIC DATA

5.2.1 Age distribution

Witvrouw *et al.* (2000) stated that patellofemoral pain syndrome (PFPS) is the most common knee complaint seen in adolescents and young children. The mean age for the sample group was 26.77 years, with the respective means for groups A, B and C being 26.50, 28.90 and 24.90 years. 50% of the subjects in this study were between the ages of 18 and 25 years. This does not correlate well with the general PFPS population, which is frequently seen in young adults with subjects being mostly between the ages of 10-20 years (Kannus *et al.*, 1999). Ninety three percent of the subjects were under 35 years of age. This correlates with the statement by Taunton *et al.* (2002) that people under the age of 34 years are at a greater risk of developing patellofemoral joint problems. Only patients between the ages of 18-45 years were recruited for this study. Although the sample group is representative of the normal PFPS population in certain aspects, future research should

consider recruiting younger subjects or alternatively subjects that are of a similar age, in order to obtain a more representative sample group.

5.2.2 Gender distribution

PFPS is significantly more common in females than in males (Salem and Powers, 2001; Taunton *et al.*, 2002). This view is supported by Davidson (1993).

The gender distribution of the entire sample was 53% male and 47% female. Group A comprised of 7 males (70%) and 3 females (30%), group B comprised of 5 males and 5 females (50%) each and group C comprised of 4 males (40%) and 6 females (60%). Group C was the most representative group and although groups A and B did not consist of significantly more females, their gender distribution was similar to previous South African studies on PFPS (Stakes, 2000; Clifton, 2003; Dippenaar 2003). The small sample group may have contributed to the disproportionate and non-normal gender distribution of the sample.

5.2.3 Activity distribution

Athletes who participate in sports involving jumping and running activities are at the greatest risk for developing patellofemoral related injuries (Salem and Powers, 2001). PFPS may however, also be present in inactive patients (Juhn, 1999).

The most common primary, secondary and tertiary activities for the treatment groups were distributed as follows:

Table 5.1 Most common activity levels.

| Activity | Group A | Group B | Group C |
|-----------------|----------------|---|----------------|
| Primary | Running | Aerobic exercise Running Gym None | Running |
| Secondary | None | None | None |
| Tertiary | None | None | None |

The three most common primary activities were running (27%), no activity and gym (17% each) and aerobic gym exercise (13%). The higher incidence of PFPS amongst runners correlates with Salem and Powers (2001), who stated that runners are amongst the group of people, who are at the greatest risk for developing PFPS. The high prevalence of inactivity amongst the sample group correlates to the comment made by Juhn (1999), who said that PFPS might present in inactive patients. The sample group therefore correlates to the normal PFPS population in terms of activity distribution.

5.3 STATISTICAL ANALYSIS

The researcher cannot accurately comment on the presence or removal of AMI due to the objective measure used in this study, as isokinetic dynamometry measures the maximum voluntary effort of a muscle and is not sensitive to small changes in motor neuron pool recruitment (Hopkins and Ingersoll, 2000).

The assumptions made in chapter one, allow for discussion of the changes in muscle strength due to changes in AMI- as a result of manipulative therapy.

5.3.1 Maximum voluntary quadriceps force output

5.3.1.1 Intra-group analysis

The mean quadriceps force scores were statistically analysed using the Wilcoxon Signed Ranks test for matched pairs. There was a significant increase in the quadriceps force after the manipulation in group B ($P = 0.05$). The quadriceps force post manipulation increased in both groups A ($P = 0.415$) and C ($P = 0.262$) but the increase did not reach a statistically significant level. Comparison of the p-values for groups A and C shows that group C improved to a greater extent.

5.3.1.2 Inter-group analysis

The Kruskal-Wallis H test was used to test for a significant difference between the treatment groups. Purposive convenience sampling was used to allocate the subjects into the treatment groups.

The mean scores, pre manipulation, were not similar between the groups. This indicates an error in the sampling procedure and allocation of the subjects. The pre manipulation mean scores should ideally be similar between the treatment groups. When the data was statistically analysed, there was no statistically significant difference in the quadriceps force between treatment groups A, B and C ($P = 0.083$).

Group B was the only group to improve significantly ($P = 0.05$) after the manipulation. The Kruskal-Wallis H test showed that there was no significant difference between the treatment groups ($P = 0.174$) post manipulation.

5.3.1.3 Discussion

The quadriceps strength increased significantly following SI joint manipulation ($P = 0.05$). Similar results were seen in studies performed by Suter *et al.* (1999) and Suter *et al.* (2000).

Both the SI and knee joints have been linked to AMI in the quadriceps muscle group (Iles *et al.*, 1990; Suter *et al.*, 1999; Hopkins and Ingersoll, 2000; Suter *et al.*, 2000). The MN pool of the quadriceps muscle originates from the L2-4 level (Moore, 1992: 387). Subject's in group C received a SI joint manipulation. The SI joint receives innervation from L2-S4. According to William (1997: 144) and Suter *et al.* (2000), altered afferent input arising from the stimulation of mechanoreceptors around the SI joint could have changed the MN pool excitability of the joint musculature. The resultant decrease in AMI would have led to an increase in the quadriceps muscle strength, in this study.

The quadriceps muscle strength was shown to increase following tibio-femoral joint manipulation and a combination of SI and tibio-femoral joint manipulations, although neither of these increases reached a statistically significant level. No other studies have investigated the effects of tibio-femoral manipulation or a combination of SI and tibio-femoral manipulations on quadriceps muscle strength. Hence, no comparison of the results following these two manipulative treatment protocols is possible.

The inter-group analysis revealed that there was no statistical difference between the treatment groups. The effectiveness of the tibio-femoral joint manipulation in stimulating the MN pool of the quadriceps muscle is questionable. Hopkins and Ingersoll (2000) recommend that when trying to reduce quadriceps AMI using TENS, the treatment area should be over the dermatomal level of the femoral nerve roots. This might alter the afferent mechanoreceptor information of the quadriceps MN pool more effectively than if the treatment area was over the knee. This however, has not been investigated.

Ranking of the P-values shows that group B ($P= 0.05$) improved the most followed by groups C ($P=0.262$) and A ($P= 0.415$). A larger and more uniform sample group may have shown more significant results.

5.3.2 The maximum voluntary hamstring force output

5.3.2.1 Intra-group analysis

The mean hamstring force values were statistically analysed using the Wilcoxon Signed Ranks test for matched pairs. Group A ($P = 0.123$) showed an improvement in the hamstring force post manipulation, but this improvement did not reach a significant level. Groups B ($P = 0.036$) and C ($P = 0.021$) both showed a significant improvement in hamstring strength post manipulation.

5.3.2.2 Inter-group analysis

The Kruskal-Wallis H test was used to test for a significant difference between the treatment groups. Purposive convenience sampling was used to allocate the subjects into the treatment groups.

Statistical analysis showed no significant difference ($P = 0.087$) between the groups pre manipulation, but the variations in the mean scores indicate slight discrepancies in hamstring strength. Post manipulation there was no significant difference between the treatment groups ($P = 0.173$).

5.3.2.3 Discussion

Hamstring weakness has been noted in PFPS patients (Clifton, 2003). There is little information in the literature about arthrogenic muscle inhibition (AMI) as it relates to the hamstring muscle.

The hamstring receives its innervation from the level of L5-S2 (Moore, 1992: 423). This again overlaps with the innervation of the SI joint (L2-S4). The role of structures within the knee joint in inhibition of the hamstring muscle is not clear.

As already discussed- according to William (1997: 144) and Suter *et al.* (2000)- altered afferent input arising from the stimulation of mechanoreceptors around the SI joint could have changed the MN pool excitability of the joint musculature. The resultant decrease in AMI would have led to an increase in the hamstring muscle strength, in this study.

Statistically, neither group was shown to be more effective than the other, which raises questions about the efficacy of the tibio-femoral joint manipulation in affecting the MN pool of the hamstring muscle.

The effect of manipulating the tibio-femoral or SI joints on hamstring strength has not been previously investigated in the literature. Therefore, comparisons of the results obtained in this study cannot be made.

The small sample group and lack of sensitivity of the maximum voluntary force measurement to detect small changes in the MN pool (which ultimately affects the force of the contraction), make it difficult to make conclusions regarding the presence and reduction of AMI in the hamstrings and inter-group comparisons of the different manipulative treatment protocols.

5.3.3 The quadriceps and hamstring ratios

5.3.3.1 Intra-group analysis

The mean quadriceps and hamstring ratios were statistically analysed using the Wilcoxon Signed Ranks test for matched pairs. All quadriceps ratios decreased post manipulation, but this decrease did not reach a statistically significant level (Group A, $P = 0.241$; Group B, $P = 0.721$; Group C, $P = 0.241$). The hamstring ratios for the different groups increased post manipulation, but this increase did not reach a significant level (Group A, $P = 0.241$; Group B, $P = 0.721$; Group C, $P = 0.241$).

The standard deviation decreased following manipulation for all groups and both muscles. This indicates that the different groups were affected similarly by the manipulations.

5.3.3.2 Inter-group analysis

The quadriceps and hamstring ratios were statistically analysed using the Kruskal-Wallis H test. Purposive convenience sampling was used to allocate the subjects into the treatment groups. Before manipulation, there was no statistical difference, with regards to the quadriceps ($P = 0.704$) and hamstring ($P = 0.704$) ratios, between the groups. This means that the quadriceps and hamstring ratios were similar between all the groups prior to manipulation.

After the manipulation was performed, there was no difference in the quadriceps ($P = 0.818$) and hamstring ($P = 0.818$) ratio between the groups.

5.3.3.3 Discussion

The different manipulative treatment protocols decreased the quadriceps and increased the hamstring ratios, but these changes did not reach a statistically significant level. All treatment groups responded similarly to the different treatment protocols, with regard to the quadriceps and hamstring ratios.

These results indicate that the different manipulative treatment protocols were not effective in altering the MN pool recruitment pattern of the quadriceps and hamstring muscles.

Pre manipulation, the quadriceps and hamstring ratios were found to be within normal limits, when compared to the study by Rosene, Fogarty and Mahaffey (2001). Hence, the MN pool recruitment pattern of the quadriceps and hamstring muscles was normal. Therefore, no significant change in the MN pool recruitment pattern would have taken place following manipulation.

The primary objective of this study was not to investigate the effect of manipulative therapy on the quadriceps and hamstring muscles. However, data obtained made analysis of the quadriceps and hamstring ratios possible. A larger sample coupled with the presence of abnormal quadriceps and hamstring ratios prior to manipulative therapy- may have resulted in more significant changes following manipulation.

5.3.5 The level of perceived pain

5.3.5.1 Intra-group analysis

The mean numerical pain rating scale (NPRS) scores were statistically analysed using the Wilcoxon Signed Ranks test for matched pairs. The NPRS for the different groups decreased post manipulation, but this decrease did not reach a significant level (Group A, $P = 0.236$; Group B, $P = 0.121$; Group C, $P = 0.271$).

5.3.5.2 Inter-group analysis

The NPRS scores were statistically analysed using the Kruskal-Wallis H test. Purposive convenience sampling was used to allocate the subjects into the treatment groups.

Before manipulation, there was no statistical difference between the groups ($P = 0.878$). This means that the level of perceived pain was similar between all the groups prior to manipulation.

After the manipulation was performed, there was no difference in the levels of perceived pain between the groups ($P = 0.729$). The descriptive statistics of the groups supports this (Group A, 31.75; Group B, 33.75; Group C, 37).

5.3.5.3 Discussion

The decrease in the NPRS scores did not reach a statistically significant level, as expected by the researcher. Following manipulation, presynaptic nociceptive inhibition of proprioceptors occurs (Lopes, 1992: 66). Closing of the gate on pain transmission (which is brought about by bursts of proprioceptive input following manipulation) is responsible for this nociceptive inhibition (Dhami and DeBoer, 1992: 121).

It is important to note that several subjects complained of an aggravation in their knee pain as a result of the isokinetic testing and this may have influenced the second numerical pain rating scale score. The aggravation of the subjects' pain by the isokinetic testing could have resulted from higher patellofemoral joint reaction (PFJR) forces associated with isokinetic knee extension testing (Nisell and Ericson, 1999). It is also not clear whether or not the manipulation itself or the isokinetic testing may have aggravated the subjects' pain, as the second NPRS score was taken immediately after the manipulative intervention, which was preceded by isokinetic testing of the thigh.

Using the Hoffman's reflex to study AMI in a pathological population would perhaps be more useful, as no physical effort is required by the subject during testing (Hopkins and Ingersoll, 2000).

5.3.6 Correlation comparisons

The Pearson's product moment correlation coefficient was used to test for a significant relationship between variables.

5.3.6.1 Correlation between the quadriceps and hamstring forces and the level of perceived pain.

No significant correlation between the quadriceps and hamstring forces and the level of perceived pain existed for any group, either before or after the relevant manipulative intervention was applied.

5.3.6.1.1 Discussion

According to the literature, a decrease in the level of perceived pain around a joint should be associated with an increase in the force of a contraction from the musculature around that joint (Lopes, 1992: 66; Dhami and DeBoer, 1992: 121; William, 1997: 144; Suter *et al.*, 2000). This was not observed during this study.

As already discussed, several subjects complained of an aggravation of their knee pain following isokinetic testing. This, along with a small and variable sample population, may have prevented a significant correlation being shown. The aggravation of the subjects' pain may have also been due to manipulation itself? The second NPRS score was taken immediately following the manipulation, which was preceded by isokinetic testing of the thigh. Therefore, it is not possible to comment on which factor (the isokinetic testing or the manipulation) precipitated the aggravation of the subjects' knee pain.

Although this study was not originally concerned with the other relationships between variables, the collection of the data made analysis possible. The following correlations were found to be significant and were therefore discussed.

5.3.6.2 Correlation between the quadriceps and hamstring forces

5.3.6.2.1 Pre manipulation

A positive correlation exists between the quadriceps and hamstring force in groups A (PC¹= 0.778; P = 0.008^{*2}), B (PC= 0.871; P = 0.001*) and C (PC= 0.842; P = 0.002*), with the strongest correlation being in group-B. This correlation shows that the hamstring force increases as the quadriceps force increases.

5.3.6.2.2 Post manipulation

A positive correlation exists between the quadriceps and hamstring force in groups A (PC= 0.886; P = 0.001*), B (PC= 0.958; 0.000*) and C (PC= 0.859; P = 0.001*), with the strongest correlation being in group-B. This correlation shows that the hamstring force increases as the quadriceps force increases.

5.3.6.2.3 Discussion

The significant correlation between the hamstring and quadriceps strengths, could be explained by reviewing the innervation of these muscles.

The quadriceps and hamstring muscles receive their innervation from the level of L2-L4 and L5-S2 respectively (Moore, 1992: 387+423). Stimulation of the mechanoreceptors around the SI joint may affect the MN pool supplied by the SI joint, L2-S4 (Cassidy and Mierau, 1992: 211-212). Stimulation of the MN pools supplied by the L2-S4 nerve roots would increase the force of contraction from these MN pools. Hence, a positive correlation between the quadriceps and hamstring strengths exists.

¹ PC = Pearson Correlation

² * = 99% confidence level

5.3.6.4 Correlations between both the quadriceps and hamstring ratios and level of perceived pain

5.3.6.4.1 Pre manipulation

There was no significant relationship between either the hamstring or quadriceps ratio and the level of perceived pain before the manipulation, for all the groups.

5.3.6.4.2 Post manipulation

A positive relationship exists between the quadriceps ratio and the level of perceived pain in group A (PC= 0.787; P = 0.007*). This means that as the quadriceps ratio increased, the level of perceived pain increased. A negative relationship exists between the hamstring ratio and the level of perceived pain in group A (PC= -0.787; P = 0.007*). This means that as the hamstring ratio increases, the level of perceived pain decreased.

5.3.6.4.3 Discussion

It is interesting to note that the relationship between both the hamstring and quadriceps ratios and the level of perceived pain, was present only after the manipulation of the tibio-femoral joint in group A. This relationship indicated that as the hamstring and quadriceps ratios increased, the level of perceived pain also increased.

This may indicate that some subjects experienced an increase in their level of perceived pain following manipulation. As already discussed, this may have been due to either the isokinetic testing or the manipulation itself. It is unclear which factor (the isokinetic testing or manipulation) precipitated the significant relationship.

CHAPTER SIX

CONCLUSIONS AND RECOMMENDATIONS

6.1 CONCLUSIONS

The purpose of this study was to investigate the effect of three manipulative treatment protocols on quadriceps muscle strength in patients with patellofemoral pain syndrome (PFPS). The first objective was to evaluate the effect of the long axis distraction manipulation of the tibio-femoral joint on quadriceps muscle strength. The second objective was to determine the effect of manipulating the sacroiliac (SI) joint on quadriceps muscle strength. The third objective was to determine the effect of manipulating both the tibio-femoral and SI joints on quadriceps muscle strength. The fourth objective was to evaluate the effect of the three manipulative treatment protocols on the subjective perception of anterior knee pain.

While this study was not originally concerned with the hamstring muscle, the concentric-concentric isokinetic testing of the thigh muscle made gathering of data possible. The effects of manipulating the tibio-femoral and/or SI joints on the hamstring was measured and quantified in a similar manner to that for the quadriceps.

The study failed to show a significant improvement in the quadriceps and hamstring muscle strengths following manipulation of the tibio-femoral joint in patients suffering from PFPS.

A significant improvement in the quadriceps and hamstring strengths was also noted following manipulation of the SI joint in patients suffering from PFPS.

A significant improvement in the hamstring force was noted after manipulation of both the tibio-femoral and SI joints. This study failed to show a significant

improvement in the quadriceps muscle strength following manipulation of both the tibio-femoral and SI joints.

Inter-group analysis revealed no difference, in terms of the treatment outcomes, between the three treatment groups.

No significant correlation was found between any of the manipulative treatment protocols (in any of the groups) and the subjective perception of anterior knee pain.

6.2 RECOMMENDATIONS

To improve the statistical significance, the following recommendations can be made:

- A larger sample size would increase the validity of the study and minimize the possibility of incorrectly accepting the null hypothesis.
- Stricter inclusion and exclusion criteria should be applied in order to get a more uniform sample group.
- The age group tested should include adolescent subjects, as PFPS is more common in this age group (Meyer *et al.*, 1990).
- Only males or females should be studied in any one study.
- The sample population should be limited to specific type of activity, or alternatively to inactive subjects.
- The sample population should exhibit uniformity in terms of the clinical presentation of PFPS and sacroiliac syndrome. Subjects should all either have unilateral or bilateral PFPS and associated sacroiliac syndrome.

Studying the reflex response (M-response and Hoffmann reflex) of a motor neuron pool using controlled electric stimulation of sensory fibres in a mixed nerve should be considered for future studies investigating the effects of treatments or interventions on arthroscopic muscle inhibition and muscle strength. This type of measurement is preferable in a pathologic population, as no effort is required by the subject during testing. The Hoffman reflex is a very sensitive measure that requires great control (Hopkins and Ingersoll, 2000). Therefore, the researcher needs to be au fait with this form of measurement if they are going to make use of it in their research studies.

After isokinetic testing, some subjects reported an aggravation of their knee pain. If isokinetic testing is to be used as an objective measuring tool, the researcher recommends that the numerical pain rating scale (NPRS) should not be used as a subjective measuring tool. The NPRS asks the subject to score their level of pain or discomfort at both extremes, when it is at its least, and worst. The average of the two scores is taken as the NPRS score (Jensen, Karoly and Braver, 1986). This method of obtaining a subjective measure needs to be reviewed if this measurement is to be taken immediately following an intervention. In this scenario, the subject needs to make an immediate decision on their perceived level of pain at the immediate time. The patient may have their perceptions altered by the immediate discomfort of the isokinetic testing. The Visual Analogue and the Verbal Rating scales are examples of pain rating scales that can rather be used to measure the amount of pain or discomfort at a specific time. Alternatively, the NPRS should be taken at another point in time.

The presence and extent of muscle inhibition in the hamstring muscle, as a result of abnormal knee or hip/ sacroiliac joint function, has not been adequately addressed in the literature. Future studies should investigate the presence and extent of muscle inhibition in the hamstring muscle as well as its relationship with MI in the quadriceps muscle. A more sensitive form of measuring MI and muscle strength would allow for further investigating the effects of spinal versus extra spinal manipulation on MI.

The researcher in this study only manipulated the sacroiliac joint. The motor neuron pool of the quadriceps and hamstring muscles is derived from the spinal levels between L2-L4 and L5-S2 respectively. Future research could be aimed at quantifying the presence and extent of spinal dysfunction between the levels of L2-L5 and its significance on MI and muscle strength in the quadriceps and hamstring muscles.

This study investigated the immediate effects of three different manipulative treatment protocols on quadriceps and hamstring strength. A study investigating the short and long term effects of the three different manipulative treatment protocols could be conducted.

Questionnaires should be designed and incorporated into the study to give it more strength in terms of subjective data.

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Do you suffer from
Knee pain

Are you aged between 18 and 45 years?

You may qualify for research being
conducted at the Durban Institute of
Technology
CHIROPRACTIC DAY CLINIC

FREE TREATMENT

is available during the study and if required
after the study has been completed

For more information contact:

Bernd Hillermann

031- 204 2205 or 031-204 2515

at the Chiropractic Day Clinic

DURBAN INSTITUTE OF TECHNOLOGY
CHIROPRACTIC DAY CLINIC
CASE HISTORY

APPENDIX B

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 History 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. xrays

- ▶ 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

DURBAN INSTITUTE OF TECHNOLOGY
CHIROPRACTIC DAY CLINIC
PHYSICAL EXAMINATION

Patient: _____ File#: _____ Date: _____

Clinician: _____ Signature: _____

Student: _____ Signature: _____

1. VITALS

Pulse rate:

Respiratory rate:

Blood pressure:

R

L

Medication if hypertensive:

Temperature:

Height:

Weight: Any change Y/N If Yes : how much gain/loss _____
Over what period _____

2. GENERAL EXAMINATION

General Impression:

Skin:

Jaundice:

Pallor:

Clubbing:

Cyanosis (Central/Peripheral):

Oedema:

Lymph nodes - Head and neck:

- Axillary:

- Epitrochlear:

- Inguinal:

Urinalysis:

3. CARDIOVASCULAR EXAMINATION

1) Is this patient in **Cardiac Failure** ?

2) Does this patient have signs of **Infective Endocarditis** ?

3) Does this patient have **Rheumatic Heart Disease** ?

Inspection

- Scars
- Chest deformity:
- Precordial bulge:
- Neck -JVP:

Palpation:

- Apex Beat (character + location):
- Right or left ventricular heave:
- Epigastric Pulsations:
- Palpable P2:
- Palpable A2:

- Pulses:**
- General Impression:
 - Radio-femoral delay:
 - Carotid:
 - Radial:
- Percussion:** - borders of heart
- Auscultation:-** heart valves (mitral, aortic, tricuspid, pulmonary)
 - Murmurs (timing, systolic/diastolic, site, radiation, grade).
- Dorsalis pedis:
 - Posterior tibial:
 - Popliteal:
 - Femoral:

4. RESPIRATORY EXAMINATION

1) Is this patient in Respiratory Distress ?

- Inspection**
- Barrel chest:
 - Pectus carinatum/cavinatum:
 - Left precordial bulge:
 - Symmetry of movement:
 - Scars:
- Palpation**
- Tracheal symmetry:
 - Tracheal tug:
 - Thyroid Gland:
 - Symmetry of movement (ant + post)
 - Tactile fremitus:
- Percussion**
- Percussion note:
 - Cardiac dullness:
 - Liver dullness:
- Auscultation**
- Normal breath sounds bilat.:
 - Adventitious sounds (crackles, wheezes, crepitations)
 - Pleural frictional rub:
 - Vocal resonance
 - Whispering pectoriloquy:
 - Bronchophony:
 - Egophony:

5. ABDOMINAL EXAMINATION

1) Is this patient in Liver Failure ?

- Inspection**
- Shape:
 - Scars:
 - Hernias:
- Palpation**
- Superficial:
 - Deep = Organomegally:
 - Masses (intra- or extramural)
 - Aorta:
- Percussion**
- Rebound tenderness:
 - Ascites:
 - Masses:
- Auscultation**
- Bowel sounds:
 - Arteries (aortic, renal, iliac, femoral, hepatic)

- **Rectal Examination**
 - Perianal skin:
 - Sphincter tone & S4 Dermatome:
 - Obvious masses:
 - Prostate:
 - Appendix:

6. G.U.T EXAMINATION

External genitalia:
 Hernias:
 Masses:
 Discharges:

7. NEUROLOGICAL EXAMINATION

Gait and Posture

- Abnormalities in gait:
- Walking on heels (L4-L5):
- Walking on toes (S1-S2):
- Rombergs test (Pronator Drift):

Higher Mental Function

- Information and Vocabulary:
- Calculating ability:
- Abstract Thinking:

G.C.S.:

- Eyes:
- Motor:
- Verbal:

Evidence of head trauma:

Evidence of Meningism:

- Neck mobility and Brudzinski's sign:
- Kernigs sign:

Cranial Nerves:

I Any loss of smell/taste:
 Nose examination:

II External examination of eye:

- Visual Acuity:
- Visual fields by confrontation:
- Pupillary light reflexes = Direct:
- = Consensual:
- Fundoscopy findings:

III Ocular Muscles:
 Eye opening strength:

IV Inferior and Medial movement of eye:

V

- a. Sensory
 - Ophthalmic:
 - Maxillary:
 - Mandibular:
- b. Motor
 - Masseter:
 - Jaw lateral movement:
- c. Reflexes
 - Corneal reflex
 - Jaw jerk

VI Lateral movement of eyes

- VII a. Motor - Raise eyebrows:
 - Frown:
 - Close eyes against resistance:
 - Show teeth:
 - Blow out cheeks:
 b. Taste - Anterior two-thirds of tongue:

- VIII General Hearing:
 Rinnes = L: R:
 Webers lateralisation:
 Vestibular function - Nystagmus:
 - Rombergs:
 - Wallenbergs:
 Otoloscope examination:

- IX & Gag reflex:
 X Uvula deviation:
 Speech quality:

- XI Shoulder lift:
 S.C.M. strength:

- XII Inspection of tongue (deviation):

Motor System:

- a. Power
 - Shoulder = Abduction & Adduction:
 = Flexion & Extension:
 - Elbow = Flexion & Extension:
 - Wrist = Flexion & Extension:
 - Forearm = Supination & Pronation:
 - Fingers = Extension (Interphalangeals & M.C.P's):
 - Thumb = Opposition:
 - Hip = Flexion & Extension:
 = Adduction & Abduction:
 - Knee = Flexion & Extension:
 - Foot = Dorsiflexion & Plantar flexion:
 = Inversion & Eversion:
 = Toe (Plantarflexion & Dorsiflexion):
- b. Tone
 - Shoulder:
 - Elbow:
 - Wrist:
 - Lower limb - Int. & Ext. rotation:
 - Knee clonus:
 - ankle clonus:
- c. Reflexes
 - Biceps:
 - Triceps:
 - Supinator:
 - Knee:
 - Ankle:
 - Abdominal:
 - Plantar:

Sensory System:

- a. Dermatomes
 - Light touch:
 - Crude touch:
 - Pain:
 - Temperature:
 - Two point discrimination:
- b. Joint position sense
 - Finger:
 - Toe:
- c. Vibration:
 - Big toe:
 - Tibial tuberosity:
 - ASIS:
 - Interphalangeal Joint:
 - Sternum:

Cerebellar function:

Obvious signs of cerebellar dysfunction:

- = Intention Tremor:
- = Nystagmus:
- = Truncal Ataxia:

Finger-nose test (Dysmetria):

Rapid alternating movements (Dysdiadochokinesia):

Heel-shin test:

Heel-toe gait:

Reflexes:

Signs of Parkinsons:

8. SPINAL EXAMINATION:(See Regional examination)

Obvious Abnormalities:

Spinous Percussion:

R.O.M:

Other:

9. BREAST EXAMINATION:

Summon female chaperon.

Inspection

- Hands rested in lap:
- Hands pressed on hips:
- Arms above head:
- Leaning forward:

Palpation

- masses:
- tenderness:
- axillary tail:
- nipple:
- regional lymph nodes:

TECHNIKON NATAL CHIROPRACTIC DAY CLINIC
REGIONAL EXAMINATION - LUMBAR SPINE AND PELVIS.

PATIENT: _____

FILE #: _____

DATE: _____

INTERN/RESIDENT: _____

SUPERVISING CLINICIAN: _____

STANDING:

Posture
Minor's Sign
Skin
Scars
Discoloration
Muscle Tone
Bony & Soft Tissue Contours

Spinous Percussion
Schober's Test (6cm)
Treadmill
Body Type
Attitude

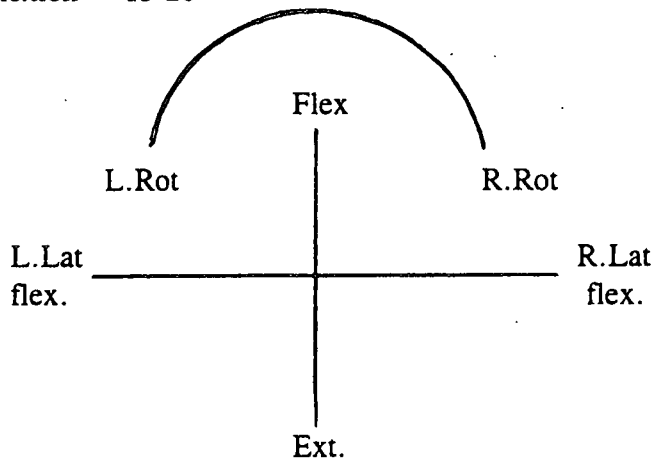
RANGE OF MOTION

Forward Flexion = 40-60° (15cm from floor)

Extension = 20-35°

L/R Rotation = 3-18°

L/R Lateral Flexion = 15-20°



SUPINE:

Skin
Hair
Nails
Palpate Abdomen/groin
Pulses (abdomen)

Observe abdomen
Fasciculations
Abdominal Reflexes

Pulses (extremities)
SLR
Bowstring
Plantar Reflex
Circumference (thigh, calf)
Leg Length:
 actual
 apparent
Sciatic Notch
Patrick FABERE
Gaenslen's Test
Gluteus Maximus Stretch
Hip Medial rotation
Psoas Test
Thomas' Test:
 hip joint
 Rectus Femoris

LATERAL RECUMBENT

S-I Compression
Ober's Test
Femoral Nerve stretch
Myotomes:
 QL
 Gluteus Medius

NON ORGANIC SIGNS

Pin Point Pain
Axial Compression
Trunk Rotation
Burn's Bench Test
Flip Test
Hoover's Test
Ankle Dorsiflexion Test.

GAIT

Rhythm
On toes (standing)
On Heels (standing)
Half squat on one leg

PRONE

Gluteal skyline
Skin rolling
Iliac crest compression
Facet joint challenge
S-I tenderness
Erichson's Test
Pheasant's Test
Myotome:
 Glut. Max
Active MF Trigger Pts:
 QL
 Glut. Med
 Glut. Min
 Glut. Max
 Piriformis
 Hamstrings
 TFL

NEUROLOGICAL EXAMINATION

| DERMATOMES | | | MYOTOMES | | | REFLEXES | | |
|------------|---|---|--------------|---|---|----------|---|---|
| | L | R | | L | R | | L | R |
| T12 | | | Hip Flex | | | Pat. | | |
| L1 | | | Hip int rot | | | Achil | | |
| L2 | | | Hip ext rot | | | H/S | | |
| L3 | | | Hip abd | | | | | |
| L4 | | | Hip add | | | | | |
| L5 | | | Knee flex | | | | | |
| S1 | | | Knee ext | | | | | |
| S2 | | | Dorsiflex | | | | | |
| S3 | | | Plantarflex | | | | | |
| | | | Eversion | | | | | |
| | | | Ext.hal.long | | | | | |

Tripod
Kemp's Test

MOTION PALPATION and JOINT PLAY:

LEFT: Upper Thoracics:
Lumbar Spine:
Sacroiliac Joint:

RIGHT: Upper Thoracics:
Lumbar Spine:
Sacroiliac Joint:

Basic Exam: Hip
Case History:

ROM: Active:
Passive:
RIM:

Orthopaedic/Neuro/
Vascular:

Observ/Palpation:

Basic Exam: Thoracic Spine
Case History:

ROM: Motion Palp:
Active:
Passive:

Orthopaedic/Neuro/
Vascular:

Observ/Palpation:

DURBAN INSTITUTE OF TECHNOLOGY

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 - 135°) _____

Extension (0 - 15°) _____

Medial Rotation (20 - 30°) _____

Lateral rotation (30 - 40°) _____

⊙ **PASSIVE MOVEMENTS**

Tissue approx _____

Bone-bone _____

Tissue stretch _____

Tissue stretch _____

Patellar movement _____

⊙ **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 _____
 "Bounce Home" _____

Anderson med-lat grind _____
 Apley's _____

⊙ **PLICA TESTS**

Mediopatellar Plica _____
 Plica "Stutter" _____

Hughston's Plica _____

⊙ **TESTS FOR SWELLING**

Brush/Stroke Test _____

Patellar Tap Test _____

⊙ **TESTS FOR PATELLA FEMORAL PAIN SYNDROME**

Clarke's Sign _____
 Waldron test _____

Passive patella tilt test _____

⊙ **OTHER TESTS**

Wilson's _____
 Fairbank's _____
 Noble Compression _____

Quadriceps Contusion Test _____
 Leg Length Discrepancy _____

⊙ **JOINT PLAY**

Movement of the tibia on the femur
 Translation of the tibia on the femur
 Long axis distraction of the tibiofemoral joint
 Inf, sup, lat, + med glide of the patella
 Movement of the inf. tibiofibular joint
 Movement of the sup. tibiofibular joint
 Movement of the sup tibiofibular joint

P → A: _____ A → P: _____
 M → L: _____ L → M: _____

 A → P _____ P → A _____
 A → P _____ P → A _____
 S → I _____ I → S _____

⊙ **PALPATION**

Tenderness _____
 Joint line _____
 Ligaments _____
 Patella: _____
 Patella tendon: _____
 Bursae: _____

Swelling _____
 Nodules/exostoses _____
 Muscles: thigh: _____
 Leg: _____
 Popliteal artery: _____

⊙ **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 | | | | |

Letter of information

Dear patient, welcome to this study.

Title of research project:

The effect of three manipulative treatment protocols on quadriceps (thigh) muscle strength in patients with Patellofemoral Pain Syndrome.

Name of supervisors: Dr. N. Gomes (031 - 572 7000)
Dr. C. Korporaal (031 - 2042611)
Mr D. Jackson (031 - 566 2165)

Name of research student: Bernd Hillermann (031 - 204 2205)

Name of institution: Durban Institute of Technology

Introduction and Purpose of the study:

This study involves research on 30 patients, to determine the effects of two manipulative treatment protocols on quadriceps (thigh) muscle strength, in people with a condition commonly known as Runners' Knee.

Procedures:

The first visit:

You will be required to undergo an initial examination at the Durban Institute of Technology Chiropractic Day Clinic. The initial consultation will include a history taking, relevant physical examination, lower back and knee regional examination and will take approximately an hour and a half.

The second visit:

The initial consultation will be followed by 1 isokinetic testing session at the Medigate Medical Centre in Umhlanga Rocks. The date of the appointment shall be made subject to availability of Mr. Jackson. The testing session involves an initial strength test of your thigh muscle. This is then followed by a treatment. Following the treatment, your thigh strength will then be retested. This visit will take approximately half an hour.

Directions to get to the Medigate Medical Centre from the Durban Institute of Technology Chiropractic Clinic and from the N2 (North Coast) are provided.

Risks/Discomfort:

The testing is low risk. This type of strength testing involves a maximum effort contraction of your thigh muscle. This may lead to some stiffness being felt in that muscle after testing.

Benefits:

There will be no charge for any of these consultations. The treatment provided is in line with normal clinical procedure for the treatment of Patellofemoral Pain Syndrome. If required, two free treatments are offered at the Chiropractic Day Clinic subsequent to the completion of your participation in the study.

New findings:

You have the right to be informed of any new findings that are made.

Reasons why you may be withdrawn from the study without your consent:

You may be removed from the study without your consent for the following reasons:

Changing any lifestyle habits, any medication or supplementation that you are on for the period of your participation in this study, as this may affect the results of the research. If you experience any discomfort during the isokinetic testing session. You are free to withdraw from the study at any time, without giving a reason.

Remuneration:

Please note that there will be no remuneration at all. Your participation in this study is voluntary.

Cost of the study:

All treatments are free of charge and your participation is voluntary.

Confidentiality:

All patient information is confidential and the results will be used for research purposes only, although supervisors and senior clinic staff may be required to inspect records.

Persons to contact for problems of questions:

You may ask questions of an independent source if you wish to my supervisors are available on the above numbers. If you are not satisfied with any area of the study, please feel free to forward any concerns to the Durban Institute of Technology Research and Ethics Committee.

Thank you for your participation in this study.

Bernd Hillermann
(Chiropractic intern)

Dr. N. Gomes
(Supervisor)

Dr. C. Korporaal
(Supervisor)

Mr. D. Jackson
(Supervisor)

Directions to get to the Medigate Medical Centre :

(A)

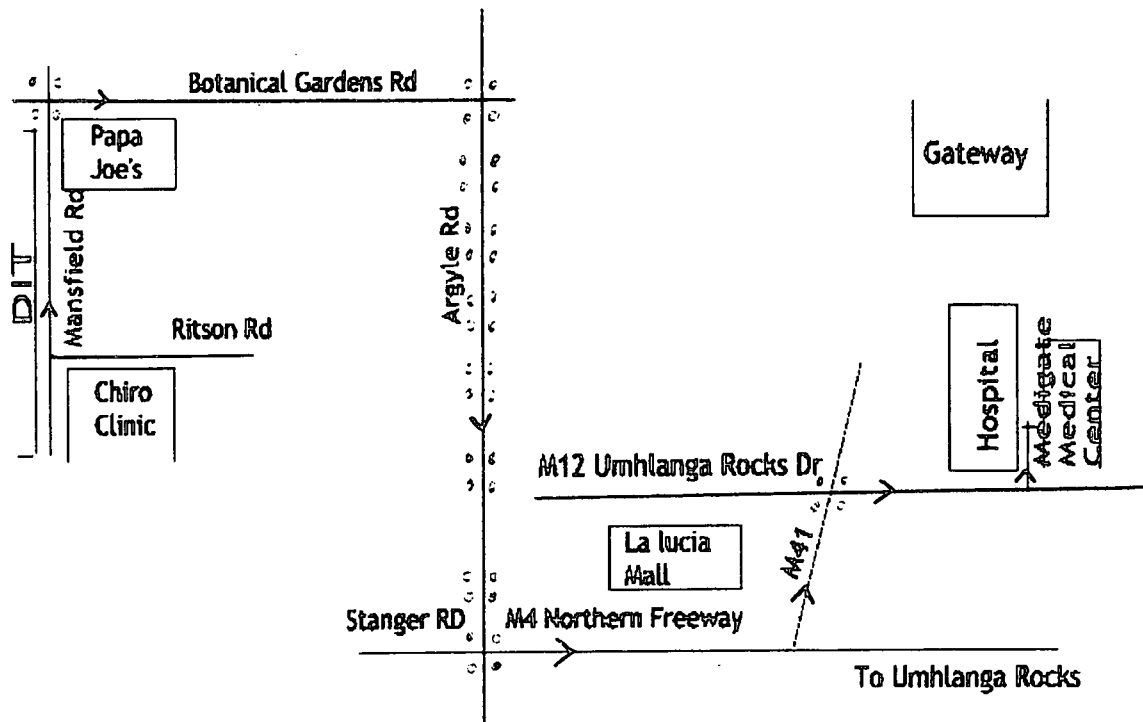
Take a right out of the Chiropractic Clinic parking lot and drive along Mansfield Road until you get to a set of robots (Papa Joe's Pizza is on your right hand side). Turn right. You are now in Botanical Gardens Road. Carry on with this road until you reach Argyle road. Turn right. Carry on going straight. At the seventh robot, turn left onto the Northern Freeway. Travel towards Umhlanga Rocks. Take the left off-ramp to the M41 which goes to Mount Edgecombe, the Gateway shopping centre and the Natal Sharks Board. This road goes up a gentle hill. Towards the top of the hill, you will take the off-ramp. Turn right into Umhlanga Rocks Drive, which will take you to Gateway and the Natal Sharks Board. Shortly after the MacDonald's (on your left hand side), you will pass the Umhlanga Hospital, and just after that you will see the Medigate Medical Centre on your left hand side. Take a left into the road between the Umhlanga Hospital and the Medigate Medical Centre. You will see GATE 1 clearly marked. Drive to this gate and push the buzzer for suite number 1.

ALTERNATIVELY:

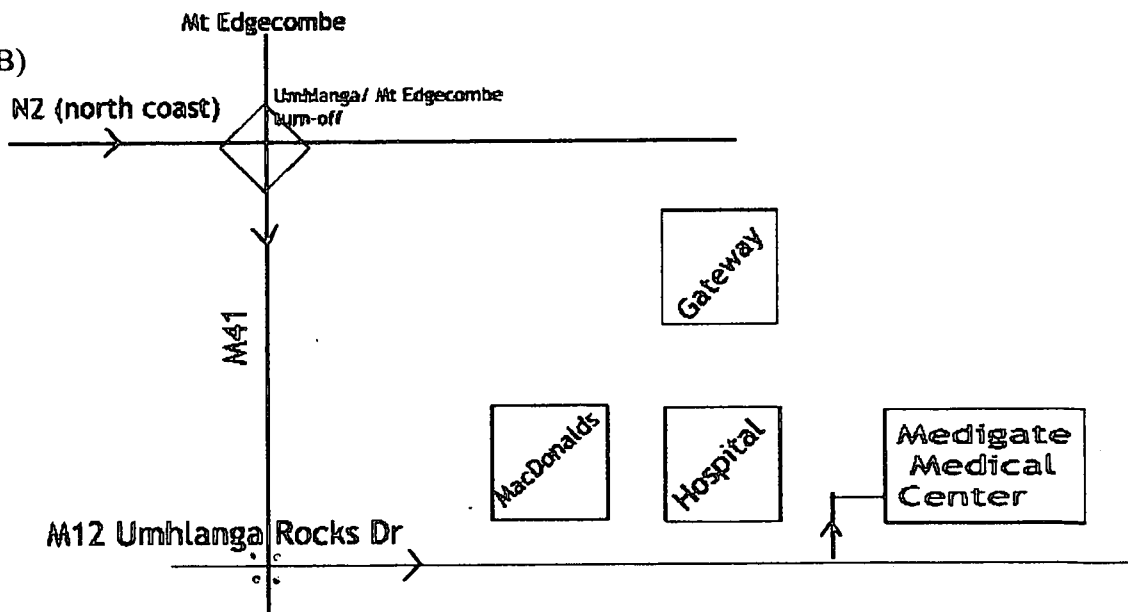
(B)

If you are traveling along the N2 (north coast) highway, take the M41 turn-off. Turn right. This takes you to the Gateway shopping centre and Umhlanga Rocks. Travel past the Gateway shopping centre (on your left). Take the second off-ramp. Turn left into Umhlanga Rocks Drive (M12). Shortly after the MacDonald's (on your left hand side), you will pass the Umhlanga Hospital, and just after that you will see the Medigate Medical Centre on your left hand side. Take a left into the road between the Umhlanga Hospital and the Medical Medicare Centre. You will see GATE 1 clearly marked. Drive to this gate and push the buzzer for suite number 1.

(A)



(B)



Key

.. = robots

NOTE: drawings not to scale

INFORMED CONSENT FORM

Date _____

Title of research project:

The effect of three manipulative treatment protocols on quadriceps (thigh) muscle strength, in patients with Patellofemoral Pain Syndrome.

Name of supervisors: Dr. N. Gomes (031-572 7000)
 Dr. C. Korporaal (031-2042611)
 Mr D. Jackson (031-566 2165)

Name of research student: Bernd Hillermann (031- 204 2205)

Name of institution: Durban Institute of Technology

This study involves research on 30 patients, to determine the effects of three manipulative treatment protocols on quadriceps (thigh) muscle strength, in people with a condition commonly known as Runners' Knee.

Please circle the appropriate answer

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

PATIENT/SUBJECT Name _____ Signature _____
 (block letters)

WITNESS Name _____ Signature _____
 (block letters)

RESEARCH STUDENT Name _____ Signature _____
 (block letters)

If you have answered NO to any of the above questions, please do not hesitate to contact my research supervisors, who will be able to assist you.