THE IMMEDIATE EFFECT OF SACROILIAC MANIPULATION ON HIP STRENGTH IN PATIENTS SUFFERING FROM CHRONIC SACROILIAC SYNDROME.

By

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DEDICATION

To my parents,
who through their example,
have given me the inspiration and determination to reach my goals.

Thank-you, I am eternally grateful.

To Jenna and Mark,
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ABSTRACT

The aim of this investigation was to investigate whether an immediate objective increase in hip strength was observed after an ipsilateral sacroiliac manipulation in patients suffering from chronic sacroiliac syndrome. The actions of hip flexion, extension, abduction and adduction were assessed.

The study also investigated the patients' subjective perception of pain due to the chronic sacroiliac syndrome before and after the manipulation.

The proposed increases in strength would have been as a result of a reduction in arthrogenic muscle inhibition. Stimulation of nociceptors caused by the chronic sacroiliac syndrome would have lead to the presence of the arthrogenic muscle inhibition within the joint.

Arthrogenic muscle inhibition has been described as an inability of a muscle group to utilise all its muscle fibres when performing a maximum voluntary contraction of that muscle group. Arthrogenic muscle inhibition is a joints natural response to pain, damage or distension within the joint. The response is an ongoing reflex inhibition of the muscles surrounding the joint in order to protect the joint.

The inhibition of the surrounding musculature clinically manifests itself as a decrease in strength of the affected muscles. The decreased strength levels hampers rehabilitation of the affected joints as active exercise forms a vital role in the rehabilitation process.

Current treatment options used to reduce arthrogenic muscle inhibition include lidocaine injection into the joint, cryotherapy and transcutaneous nerve stimulation. These treatments are aimed rather at the reduction of pain, joint effusions and atrophy of the related musculature than at the reduction of arthrogenic muscle inhibition.

Recent studies have proposed that manipulation reduced arthrogenic muscle inhibition by causing excitation of the joint receptors, called the Wyke receptors. Stimulation of these joint receptors is thought to cause an alteration in the afferent input to the motorneuron pool resulting in a reduction of arthrogenic muscle inhibition.

This study aimed to investigate whether sacroiliac manipulation could reduce arthrogenic muscle inhibition at the hip by assessing the immediate gains in hip muscle strength.

The problem statement was to evaluate if an immediate subjective or objective change in hip strength was observed after an ipsilateral sacroiliac manipulation in patients suffering from chronic sacroiliac syndrome.

The Null-hypothesis for the study was that there was no difference in the comparison of observations from pre- manipulation to post-manipulation. The Alternate hypothesis stated that there was an increase between the observations when comparing the pre-manipulation values to post-manipulation values.

The sample consisted of thirty male patients that were diagnosed with chronic sacroiliac syndrome. The diagnosis of chronic sacroiliac syndrome was confirmed at an initial screening appointment at the Durban Institute of Technology Chiropractic Day Clinic. This study was a prospective randomised pre- post- investigation, as the subject's hip strength was compared to himself before and after the manipulation.

Objective assessments were conducted at a second appointment by means of isokinetic testing on an Orthotron II Isokinetic Rehabilitation System. The isokinetic assessments were performed by a registered biokineticist. Isokinetic hip strength readings were performed immediately before and after the

sacroiliac manipulation. The sacroiliac manipulation was only administered to the subjects at the second appointment.

Subjective assessments were by means of the numerical pain rating scale 101. This tool was used to evaluate the subjects' perceived pain. The subject's were asked to evaluate their pain at their first consultation (screening appointment), this was taken as subjective reading one. The second subjective reading was taken immediately after the manipulation and second objective reading (isokinetic assessment) at the second appointment.

The data was then analysed through the use of the SPSS statistical package. Inter-group comparisons were drawn using the ANOVA test to be followed by paired T-tests if the ANOVA result was significant. Intra-group comparisons were drawn using the paired T-tests. Descriptive statistics were drawn using means and percentages of the data in graphs and tables.

Inter-group analysis drawn by the ANOVA test produced a statistically insignificant result. This indicated that all the actions responded to the manipulation in the similar trend. Not one action responded significantly superior or inferior when compared to the other actions' responses. Due to the statistically insignificant result of the ANOVA test the follow on paired T-tests were deemed unnecessary.

Intra-group analysis assessed the response of each individual action to the manipulation. All actions showed an increase in strength after the manipulation. The results for flexion, abduction and adduction were statistically significant, the result for extension was however statistically insignificant.

This study suggests that the presence of arthrogenic muscle inhibition at the hip, is a result of a chronic sacroiliac syndrome. This study further supports the use of manipulation in the treatment of arthrogenic muscle inhibition.

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Hip Flexion is the anterior bending of the femur in the saggital[1] plane (Moore and Dalley, 1999:6).

Reid, (1992:604) described the range of motion of hip flexion to be approximately 140°. The motion is limited by the soft tissue apposition of the thigh on the abdomen. Hip flexion is diminished to approximately 90° when the knee is placed in the extended position. This is due to an increase in hamstring tension.

2. Extension

Extension is the action of straightening or increasing the hip angle in the posterior direction along the saggital plane around the X-axis (Moore and Dalley, 1999:8).

Only about 10° to 20° of true extension occurs at the hip. The illiofemoral and ischiofemoral ligaments and the Iliopsoas muscles limit this movement to such a narrow range (Reid, 1992:607).

3. Abduction

Hip abduction involves the movement of the femur away from the median plane. Movement is in the coronal plane and around the Z-axis (Moore and Dalley, 1999:8).

The action of hip abduction is limited by muscle tightness in the adductor muscles and via the pubofemoral ligament and medial aspect of the illiofemoral ligament. The range of motion is approximately 50°,

[1] An imaginary plane that divides the body into left and right portions (Moore and Dalley, 1999:3)

until the pelvis begins to tilt to prevent impingement of the greater trochanter. However, anatomically the joint can allow up to 90° of motion, which can be achieved by abducting the leg when it is in the laterally rotated position (Reid, 1992:608).

4. Adduction

Adduction is the movement of the femur towards the median plane (Moore and Daley, 1999:8).

The opposite leg limits hip adduction. With the contra-lateral leg flexed, 40° of adduction is possible. This motion is limited by the lateral band of the illiofemoral ligament and the ligament of the head of the femur (Reid, 1992:609).

5. Internal and External Rotation

The actions of internal and external rotation can be performed at the hip (Moore,1992:469). Both these actions were not included in this study as both actions could not be performed on the Cybex Dynamometer utilised in this study (Jackson, 2003).

CHAPTER ONE

1.1. INTRODUCTION

Arthrogenic muscle inhibition has been described as an inability of a muscle group to utilise all its muscle fibres when performing a maximum voluntary contraction of that muscle group (Suter, *et al.* 2000). Arthrogenic muscle inhibition is a joints natural response to pain, damage or distension within the joint. The response is an ongoing reflex inhibition of the muscles surrounding the joint in order to protect the joint (Hopkins and Ingersoll, 2000).

Arthrogenic muscle inhibition is caused by the stimulation of joint receptors'[1] to pain, ligament stretching, capsule compression, effusion or irritation of the joint due to injury within the joint (Spencer, Hayes and Alexander, 1984). Stimulation of the joint receptors causes excitation of interneurons[2] (Hopkins and Ingersoll, 2000), which transmit excitatory or inhibitory impulses (Crossman and Neary, 1995).

Joint receptors appear to stimulate inhibitory interneurons causing an inhibition of the joints' motorneuron pool[3], causing a reduction in the motorneuron recruitment (Hopkins and Ingersoll, 2000). The reduction in motorneuron recruitment is seen clinically as a reduction in strength of the effected muscle group (Suter, *et al.* 1999).

[1] Joint receptors are specialised cells or sub-cellular structures that change their properties in response to specific stimuli of various types (Hopkins and Ingersoll, 2000).
[2] Interneurons are small, highly excitable neural cells with many interconnections, many directly innervate the anterior motor neurons (Guyton and Hall, 1997:441).
[3] The complete innervation to one muscle group (Hopkins and Ingersoll, 2000).

The decrease in strength caused by muscle inhibition hampers rehabilitation of the affected joints as active exercise forms a vital role in the rehabilitation process (Hopkins and Ingersoll, 2000 and Suter, *et al.* 1999).

Current treatment options used to reduce arthrogenic muscle inhibition include lidocaine injection into the joint, cryotherapy and transcutaneous nerve stimulation (Hopkins and Ingersoll, 2000). These treatments are aimed rather at the reduction of pain, joint effusions and atrophy of the related musculature than at the reduction of arthrogenic muscle inhibition (Ingersoll, Palmieri and Hopkins, 2003).

Suter, *et al.* (1999) and Suter, *et al.* (2000) proposed that the effects of sacroiliac manipulation[4] reduced arthrogenic muscle inhibition. A manipulation is believed to cause excitation of joint receptors, called the Wyke receptors (Leach, 1994:63). Stimulation of these joint receptors causes an alteration in the afferent[5] input to the motorneuron pool resulting in a reduction of arthrogenic muscle inhibition (Suter, *et al.* 2000).

A symptomatic sacroiliac syndrome is characterised by amongst other signs and symptoms: joint dysfunction[6] in the sacroiliac joint and pain over the sacroiliac joint (McCulloch and Transfeldt, 1997) and therefore sacroiliac syndrome is considered to be a large contributor to low back pain (Suter *et al.* 2000). The sacroiliac joint is richly innervated with nociceptive[7] mechanorecptors[8] making it plausible that this joint is a significant source of low back pain (Sakamoto *et al.* 2001). A symptomatic sacroiliac joint will cause stimulation of the nociceptors in and associated with the joint (Sakamoto, *et al.* 2001).

[4] A passive manual manoeuvre during which a joint is quickly brought beyond its restricted physiologic range of movement and beyond its elastic barrier without exceeding the boundaries of anatomic integrity (Redwood, 1997:339).
[5] The sensory function of neural elements (Redwood, 1997:333).
[6] (Joint Restriction) the temporary immobilisation of a joint in a position that it may normally occupy during any phase of normal movement (Redwood, 1997:338).
[7] Pain receptors, which detect damage occurring in the tissues, whether physical, mechanical or chemical damage (Guyton and Hall 1997: 376).
[8] A receptor that is excited by mechanical pressures or distortions, as those responding to sound, touch and movement (Redwood, 1997:339).

A sacroiliac syndrome causing stimulation of the nociceptors and mechanoreceptors could lead to the development of arthrogenic muscle inhibition. Arthrogenic muscle inhibition arising from the sacroiliac joint would lead to inhibition of the muscles that fall within the motorneuron pool of the sacroiliac joint.

With the sacroiliac joint being innervated by L2-S2 (Suter, *et al.* 2000), The motorneuron pool of the hip joint in the actions of flexion, extension, adduction and abduction fall into the same segmental nerve supply as the sacroiliac joint (Moore and Dalley, 1999:540).

Based on the above information it is of the researchers opinion that stimulation of the sacroiliac joints' nociceptors by a symptomatic sacroiliac syndrome can lead to arthrogenic muscle inhibition of the motorneuron pool responsible for actions at the hip and manipulation of the sacroiliac joint could reduce this inhibition.

1.2. THE AIM

The purpose of the study was to demonstrate if there was an immediate change in hip strength after an ipsilateral sacroiliac manipulation and to quantify that change if it was shown to exist.

Therefore the aims of the investigation were to:

1. Evaluate if male subjects, suffering from chronic sacroiliac syndrome, demonstrated an objective change in their hip strength in the actions of flexion, extension, adduction and abduction immediately after an ipsilateral[9] sacroiliac manipulation. The objective measurements were taken utilising the Cybex Orthotron II Isokinetic Rehabilitation System.

2. Evaluate if male subjects, suffering from chronic sacroiliac syndrome, demonstrated a change in their subjective perception of pain immediately after an ipsilateral sacroiliac manipulation. The subjective data was to be taken using the Numerical Pain Rating Scale.

This data was then to be statistically analysed using the SPSS package.

[9] Relating to the same side (Crossman and Neary, 1995:162)

1.3. ASSUMPTIONS

1. Arthrogenic muscle inhibition manifests itself as the inability of a muscle to utilise all its muscle fibres to their maximum during a voluntary effort. Clinically this is seen as a decrease in the strength of the affected muscle (Suter, *et al.* 2000).

2. Arthrogenic muscle inhibition is primarily caused by stimulation of mechanoreceptors in a joint, causing altered afferent innervation of the motorneuron pool (Hopkins and Ingersoll, 2000). Based on the above explanation, stimulation of nociceptive mechanoreceptors (Sakamoto, *et al.* 2001) in a sacroiliac syndrome can lead to muscle inhibition within the sacroiliac joints motorneuron pool.

3. Spinal manipulation has been proposed to stimulate mechanoreceptors and proprioceptors[10] in and around the manipulated joint. This stimulation causes an altered afferent input from the mechanoreceptors causing changes in the excitability of the manipulated joint's motor neuron pool (Suter, *et al.* 1999 and Suter, *et al.* 2000).

[10] Joint receptors that transmit information on proprioception, which is the detection of position and movement of body parts (Crossman and Neary, 1995,162).

1.5 POTENTIAL BENEFITS OF THE STUDY

1. Suter, *et al.* (1999) and Suter, *et al.* (2000) showed that sacroiliac manipulation increased the strength of muscles affected by arthrogenic muscle inhibition that were within the manipulated joints' motorneuron pool. They proposed that the observed increases in strength were caused by the effect of reducing arthrogenic muscle inhibition. The immediate effects of sacroiliac manipulation on hip muscle strength in patients suffering from chronic sacroiliac syndrome have not been investigated. This study aims to add to the limited knowledge available on the treatment of arthrogenic muscle inhibition.

2. This study could provide a rationale for the inclusion of sacroiliac manipulation in the rehabilitation protocol of the hip joint when patients present with decreased hip strength as a result of arthrogenic muscle inhibition. Active exercise forms an important role in the rehabilitation process yet the gains from active exercise are diminished in the presence of arthrogenic muscle inhibition (Hopkins and Ingersoll, 2000). If ipsilateral sacroiliac manipulation immediately increases hip strength then sacroiliac manipulation can prove a beneficial therapeutic tool in the rehabilitation process.

3. The knee has been the focus of most of the recent studies on arthrogenic muscle inhibition Suter, *et al.* (1999); Suter, *et al.* (2000); Hopkins, *et al.* (2000) and Clifton (2003). Little focus has been placed on the effects of arthrogenic muscle inhibition at the hip (Arokoski, *et al.* 2002). This research aims to investigate the effects of arthrogenic muscle inhibition at the hip.

CHAPTER TWO

2. REVIEW OF THE RELATED LITERATURE

2.1 INTRODUCTION

The following chapter aims to create a clear understanding regarding the definition, natural history, treatment and the neurophysiology of arthrogenic muscle inhibition. Further importance will be dedicated to the anatomy of the hip and sacroiliac joints, sacroiliac syndrome and isokinetic muscle testing.

2.2. THE SACROILIAC JOINT

2.2.1. Anatomy

Walker (1986) stated that the sacroiliac joint is partly a synovial[1] joint and partly a syndesmosis[2].

The sacroiliac joint is formed by the articulation of the sacrum with the ilium, and therefore the joints are located bilaterally at the base of the spine. The joint has limited movement, which contributes to its strength. This strength is necessary as the joint transmits weight from the vertebral column to the lower limbs (Hendler *et al.*, 1995).

Harrison, Harrison and Troyanovich (1997) described the sacrum as being wedge shaped with the base orientated anterior and superior. Compression due to gravity and the weight of the trunk forces the sacrum to move tightly between the innominate bones. Resulting in the sacrum

[1] Synovial Joints are the most common type of joints and are characterised by a joint cavity, articular cartilage and articular capsule (Moore, 1992:16)
[2] A syndesmosis is a type of fibrous joint in which the intervening fibrous connective tissue forms an interosseous membrane or ligament (Magee, 2002:570).

being positioned with the base down and forward and the inferior aspect positioned posterior and superior. The authors (Harrison, Harrison and Troyanovich, 1997) compared the sacrum to fulfilling the role of a keystone in the pelvic ring.

Posteriorly the upper two thirds of the joint is covered by the ilium, the lower third is covered by sacroiliac ligaments (Hendler *et al.*, 1995).

The articular surfaces of the sacroiliac joint are covered by cartilage. The iliac surface is covered by thin fibro-cartilage and the articular surface of the sacrum is covered by hyaline cartilage (Kirkaldy-Willis 1992:71).

Anatomical variations in the sacroiliac joint occur between the sexes.

Table 2.1. Sex differences between the sacroiliac joints

	MALES	FEMALES
Surface texture	Increased roughness of surfaces	Smoother joint surfaces. Even observed in advanced age
Size	Larger	Smaller
Orientation	Not as flat	Flatter
Movement	Less movement	Increased movement, possibly due to childbearing.

(Harrison, Harrison and Troyanovich, 1997)

2.2.2. Sacroiliac joint ligaments

The sacroiliac ligaments contribute to the stability of the joint (Hendler *et al.* 1995). Harrison, Harrison and Troyanovich (1997) proposed that the ligaments functioned to limit every possible motion and oppose strong forces for prolonged periods of time and are therefore accordingly adapted.

The following sacroiliac ligaments will be discussed in further detail: anterior sacroiliac ligament, posterior sacroiliac ligament, interosseous sacroiliac ligament, iliolumbar ligament, sacrotuberous ligament and the sacrospinous ligament

2.2.2.1. Anterior sacroiliac ligament

The thickening of the anterior inferior joint capsule forms this ligament. It is well developed near the arcuate line[3] and the posterior superior iliac spine (Harrison, Harrison and Troyanovich, 1997).

Hendler *et al.* (1995) described the ligament as being thin, easily distended by intra-articular swelling and palpable on rectal examination.

The function of the ligament is to oppose translation[4] of the sacrum up or down and separation of the joint surfaces (Harrison, Harrison and Troyanovich, 1997).

[3] A radiographical analysis of the sacral foramen assessing for sacral fractures (Yochum and Rowe, 1996:705).

2.2.2.2. Posterior sacroiliac ligament

The thickness and strength of this ligament protects the sacroiliac joint from violent trauma to such an extent that trauma to the joint may cause fracture on either side of the joint rather than dislocation of the joint (Hendler, *et al.* 1995).

2.2.2.3. Interosseous sacroiliac ligament

Harrison, Harrison and Troyanovich (1997) described this ligament as a massive ligament filling the irregular spaces superior and posterior to the sacroiliac joint. The interosseous ligament was described as the largest syndesmosis of the body and the strongest connection in the sacroiliac region.

The same authors (Harrison, Harrison and Troyanovich, 1997) speculated that the function of this ligament was to resist joint separation and translation along the X and Z axes i.e. in the vertical and anteroposterior movements respectively.

2.2.2.4. Iliolumbar ligament

The iliolumbar ligament connects the illiac crest to the transverse process of the fourth and fifth lumbar vertebra (Hendler *et al.* 1995). Its function is to limit all movements between the sacrum and the distal lumbar spine, and prevents translation of the sacrum out of the pelvic girdle and separation of the ilia from the sacrum (Harrison, Harrison and Troyanovich, 1997).

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[4] Motion of a rigid body in which a straight line in the body always remains parallel to itself (White and Panjabi, 1990:688).

2.2.2.5. Sacrotuberous ligament

The sacrotuberous ligament opposes sacral rotation during flexion (around the X-axis) due to its attachment to the sacrum and the ischium (Harrison, Harrison and Troyanovich, 1997).

2.2.2.6. Sacrospinous ligament

The sacrospinous ligament is a thin triangular shaped ligament that attaches the sacrum to the ischium. It prevents rotation of the sacrum around the X and Y axes (Harrison, Harrison and Troyanovich, 1997).

2.2.3. Developmental anatomy

Synovial joints usually develop between two primary growth centres, which lead to the development of a cartilaginous growth model. The joint matures by ossification of these cartilage growth models until ultimately a synovial joint develops (Bernard, 1997:73).

However, the development of the sacroiliac joint is different as it develops between a hyaline cartilage model and a newly ossified ilium. This is significant as it provides an explanation into the reason for unequal chondrogenesis, which will explain the variation in cartilage between the two joint surfaces (Cassidy, 1994:24).

Gradual ossification of the cartilaginous regions, which separates the sacral vertebrae from the pelvic bones, occurs up to the age of eighteen. Synostosis[5] occurs at the age of eighteen and is completed by about the

[5] The bony fusion between 2 adjacent bones that are closely opposed (Yochum and Rowe,1996:663).

twenty-fifth year when the joint will have acquired its adult morphology (Gotz, 1993:132).

Before the fourth decade the joint undergoes thinning of the cartilaginous elements, after which marginal ankylosis may become evident. This process continues until approximately the eighth decade when most individuals reach complete bony ankylosis of the joint causing a loss of mobility within the sacroiliac joint (Mior, Ro and Lawrence, 1999:214).

2.2.4. The surface texture

The joint surfaces are auricular shaped and are covered with ridges and depressions (Indahl, 1999).

These ridges and depressions were found to vary in height, number and orientation. However the ridges where found to be reciprocal to the depressions; i.e. an elevation of the sacral surface fits a depression of the iliac surface and vice versa. The height and depth of the ridges and depressions were found to vary from 2 to 11 mm (Harrison, Harrison and Troyanovich, 1997: 608). These authors concluded that the above ridges and depressions were non-pathological and were present to help the joint adapt to the stress of weight bearing and add to the stability of the joint by reducing the mobility in the joint.

Males were found to have more ridges and depressions than females, this is thought to be due to childbearing and a difference in the centre of gravity in females (Hendler *et al.* 1995).

Later in life the joint develops fibrous adhesions resulting in gradual obliteration of the joint space. This process continues until late in life when the joint is completely fibrosed. The joint surfaces contribute to the strength of the joint at the expense of the movement within the joint (Hendler *et al.* 1995).

2.2.5. Movement at the sacroiliac joint

The sacroiliac joint differs from most synovial joints in that they possess very little mobility. The lack of movement at the joint results in the joint being very stable, hence the joints responsibility for transmitting the weight of most of the body to the hip bones. (Moore and Dalley, 1999: 518)

During flexion and extension of the trunk there is a small amount of anterior to posterior rotary movement around the transverse axis (Hendler, *et al.* 1995). Vleeming *et al.* (1990) stated that the sacroiliac joint's main movement was in the action of nutation[6] and contra-nutation[7].

Indahl *et al.* (1999) in a study on porcine subjects showed that the sacroiliac joint moved 0.5 to 1.6mm in translation and up to 4° in rotation.

The sacroiliac joint has been found to be able to withstand large compression and bending movements, but is vulnerable to shear forces on account of their relatively flat surfaces (Snijders, Vleeming and Stoeckart, 1993).

Hendler *et al.* (1995) proposed that the greatest change in position of the sacrum in relation to the ilium results when the individual moves from the recumbent to the standing position. The sacral promontory[8] was found to move forward 5 to 6 mm due to the body weight being loaded on the sacrum.

[6] Nutation is the forward motion of the base of the sacrum into the pelvis or the backward rotation of the ilium on the sacrum (Magee, 2002:570).
[7] Contra-nutation is opposite to nutation, it indicates an anterior rotation of the ilium on the sacrum (Magee, 2002:570).
[8] The anterior projection of the first sacral vertebra (Moore and Dalley, 332:1999)

2.2.6 Biomechanics and function

Harrison, Harrison and Troyanovich, (1997) described the sacroiliac joint as being responsible for the transmission of forces from the spine to the lower extremities and the dampening and distribution of ground reactive forces when walking. The sacroiliac joint is adequately suited to perform this function.

A biomechanical model that was proposed by Vleeming *et al.* (1990) explained that the stability of the sacroiliac joint relied on a high friction co-efficient between the joint surfaces and a large wedge angle.

The ridges and depressions on the articular surface's of the sacroiliac joint causes a rough texture which is responsible for the high friction co-efficient. The depressions have been found to be more important in the development of friction than the ridges (Vleeming *et al.*, 1990).

The findings of Vleeming *et al.* (1990) are in keeping with Snijders, Vleeming and Stoeckart (1993) who concurred that the sacroiliac joint is responsible for the transfer of large lumbosacral loads to the iliac bones and legs, which is only possible in the presence of a stable pelvic arch. The sacroiliac joint has relatively flat surfaces, which allow the transfer of large moments of force, however this causes the joint to be vulnerable to transverse forces near the joint (Snijders, Vleeming and Stoeckart, 1993).

A sacroiliac joint compression theory has been formulated that suggests the transverse forces are counteracted by compression of the sacroiliac joints. This compression is caused by a self-bracing mechanism, which is formed partly by the muscles and ligaments that support the joint being orientated perpendicularly to the joint surfaces and partly by the loading mode of an arch. Therefore the self-bracing mechanism is influenced by friction, the wedge angle of the joint, joint compression and the geometry of an arch. The efficiency of the self bracing mechanism is reduced in the

presence of low muscle function, i.e. sedentary people, and in the presence of ligamentous laxity i.e. during pregnancy (Snijders, Vleeming, Stoeckart, 1993).

The above theory holds true when the person is standing upright and equal loads are placed on both legs, in this situation the suprasacral load is approximately 60% of the body weight and this load is then transferred to the sacrum (Snijders, Vleeming and Stoeckart, 1993).

The biomechanical principle of the sacroiliac joint allows for compression and bending of the joint but avoids shear movements. The resistance to sliding of the joint is caused by

1. Roughened joint surfaces causing an increase in the friction co-efficient.
2. Loading due to high compression.
3. Development of groves and ridges.

(Snijders, Vleeming and Stoeckart, 1993)

2.2.7. Muscles of the sacroiliac joint

Mior, Ro and Lawrence (1999:216) divided the muscles of the sacroiliac joint into three main groups:

Table 2.2. Muscles of the sacroiliac joint

Muscles	Causing movement of	Resultant sacroiliac motion
Erector spinae, Multifidus, Rectus abdominus.	Vertebral column	Sacral motion
Iliopsoas, Gluteus maximus, Piriformis, Hamstrings Sartorius.	Ipsilateral thigh	Iliac motion
	Pelvis	Iliac motion
Erector spinae, Rectus abdominus	Anterior/posterior	
Erector spinae, Multifidus	Lateral movement	

2. 2.8. Innervation

The sacroiliac joint and capsule has a complex innervation that provides pressure and position sense to the central nervous system (Ombregt, *et al.* 1995:691). Pain and temperature have been found to be transmitted via numerous unmyelinated[9] free nerve endings found within the joint (Mooney, 1997:41).

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Maitland, *et al.* (2001:384) suggested that the sacroiliac joint received a diverse and wide innervation from the nerve levels of L2 to S4. They proposed that the innervation of the joint was responsible for the inconsistent and variable presentation of sacroiliac joint pain patterns.

Indahl, *et al.* (1999) reported the similar segmental nerve supply of the sacroiliac joint as Maitland, *et al.* (2001). They found that the sacroiliac joint was richly innervated. The main supply was predominantly from L4 to S1 nerve roots and to a lesser extent by S2 and L3 nerve roots. Similarly, Suter, *et al.* (2000) stated that the nerve supply of the anterior sacroiliac joint was supplied by the anterior primary divisions of L2 through S2, which project onto the main lower limb nerves i.e. the femoral and tibial nerves.

Murata, *et al.*, (2001) elaborated on the innervation of the sacroiliac joint by adding that the joint is mainly innervated by the dorsal root ganglia (DRG) of L1 to S2. They added that the dorsal aspect of the joint is innervated by the dorsal rami of the DRG of L4 to S2 and the ventral aspect of the joint is innervated by the DRG of L1 to L3. The innervation of the ventral aspect (L1 to L3) of the joint pass through the sympathetic trunk.

The sacroiliac joint has thick, thin and unmyelinated nerve fibres which are associated with a wide range of sensory receptors, including encapsulated mechanoreceptors, which make it plausible that the sacroiliac joint posses a wide range of these sensory receptors (Indahl, *et al.* 1999).

[9] The nerve is not encased in a myelin sheath (Guyton and Hall, 1997:55)

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 system work together to gather, transmit and process information from many different neurophysiological systems in order to co-ordinate movement (Hopkins and Ingersoll, 2000). A review of the related neuroanatomy is necessary in order to gain a full understanding into arthrogenic muscle inhibition.

The next few paragraphs aim to look into the neurophysiology of the development of arthrogenic muscle inhibition at all its stages. From the joint, joint receptors, transmission to the spinal cord, interneurons, the transmission of signals to supra-spinal structures and the descending tracts to the motor neurons.

2.3.2. The Joint

Co-ordinated movement requires the integration and processing of large amounts of neurological information. A lot of the information to achieve motion is received from the joints themselves, which transmit electrical impulses to supra-spinal structures concerning the environment, position and movement of the joint (Levangie and Norkin, 2001:69). The joint is able to obtain this information from joint receptors that are located in joint capsules, ligaments and tendons (Levangie and Norkin, 2001:71). This information reaches the spinal cord and is believed to be the influential factor associated with arthrogenic muscle inhibition (Hopkins, *et al.* 2000).

2.3.3. The joint receptors

Joint receptors are specialised sensory nerve endings that respond to mechanical, thermal or chemical stimulation (Hopkins and Ingersoll, 2000). Stimulation of these receptors results in the transmission of an action potential along the sensory nerve to which they are attached, ultimately resulting in the conduction of an electrical impulse to the central nervous system (Crossman and Neary, 1995:23).

Joint receptors have a proprioceptive and a mechanoreceptive function, the mechanoreceptive function can be noxious or non-noxious (Hopkins, Ingersoll and Palmieri, 2003). This means that they play a role in:

1) joint position sense and body configuration (proprioceptive)
2) Initiating protective reflex mechanisms and stabilisation of the joint.

(Hopkins and Ingersoll, 2000)

Ingersoll, Palmieri and Hopkins (2003) found that arthrogenic muscle inhibition was mainly caused by the stimulation of **mechanoreceptors,** to a lesser degree by **free nerve endings** and **specialised nociceptors**. All of which are types of joint receptors.

Free nerve endings are pain receptors (Darby and Daley, 1995:253) that were described by Hopkins and Ingersoll (2000) as being non-specialised, non-encapsulated and unmyelinated or finely myelinated[10] and play a role in the crude perception of initial movement.

> **Nociceptors** are pain receptors and have been classed into 3 groups: mechanical, thermal and polymodal groups (Kingsley, 1996:130). Mechanical and thermal nociceptors are stimulated by mechanical and temperature stimuli respectively. The

[10] The axon of the nerve is surrounded by a myelin sheath, which is formed by schwann cells. The myelin sheath helps with nerve conduction (Guyton and Hall, 1997:55).

polymodal nociceptors are activated by mechanical, chemical or temperature stimuli (Jacobs and Lowe, 1999:78). Sakamoto *et al.* (2001) in a study on porcine joint receptors concluded that receptors innervated by nociceptive sensory nerve fibres (i.e. type A-delta, type C and type IV fibres - to be discussed later) were nociceptors.

Mechanoreceptors respond to mechanical pressures or distortion in the joint (Redwood, 1997:339) and Hopkins and Ingersoll (2000) outlined three different types of mechanoreceptors, they are:

1) Ruffini endings

These were described as slow adapting receptors with a low threshold. This means that they respond to the slightest stimuli but because they are slow adapting they are suited for prolonged discharge. These receptors are often located in the joint capsule so they respond to changes in capsular pressure, often associated with joint effusions. Ruffini endings also give information on joint limitations and proximity (Hopkins and Ingersoll, 2000).

2) Pacinian corpuscles

These dynamic mechanoreceptors are found mainly in the fibrous periosteum near articular attachments (Jones, 1999:119). They respond quickly to stimuli, therefore any movement causes stimulation of these receptors (Hopkins and Ingersoll, 2000). They are active during acceleration and deceleration and are inactive in immobile joints and joints that are moving at a constant velocity (Freiwald, Reuter and Engelhardt, 1999:83).

3) Golgi-like bodies

These receptors resemble tendon organs and are commonly found in ligaments around the joint (Jones, 1999:119). During the initiation of movement they fire rapidly, they then reduce to a slow steady discharge. These receptors play an important role in joint position sense (Freiwald, Reuter and Engelhardt, 1999:83-84).

Activation of a joint receptor by mechanical, thermal or chemical stimuli causes a change in the membrane potential (depolarization) of the sensory nerve that innervates the stimulated sensory nerve (Guyton and Hall, 1997: 55). When the depolarization crosses the threshold of the sensory nerve an action potential[11] is developed. The action potential travels along the axon[12] of the sensory nerve towards the spinal cord (Guyton and Hall 1997:50).

2.3.4. Transmission to the spinal cord

Information is transmitted to the spinal cord via sensory (afferent) nerves (Crossman and Neary, 1995). It is necessary to classify the different types of sensory (afferent) nerves that exist. These nerves carry the action potential to the spinal cord (Crossman and Neary, 1995).

[11] An action potential is a rapid change in the membrane potential of the nerve, causing the transmission of a nerve signal (Guyton and Hall, 1997:50).

2.3.5. Classification of sensory nerve fibres.

Different methods exist in order to classify sensory (afferent) nerve fibres. We will look at two of these classifications. The one classification is based on the diameter of the nerve fibre. The other classification is based on the origin, function and conduction velocity of the nerve.

1) Classification according to nerve diameter

Table 2.3. Sensory nerve classification by diameter

Fibre type	Fibre diameter	Myelination	Functions
Type a			
Beta (β)	5 –12	Myelinated	Sensory, touch, pressure and vibration
Delta (δ)	2-5	Thinly myelinated	Sharp localised pain, temperature, touch
Type B	<3	Myelinated	Preganglionic, autonomic
Type C	0.4 – 1.2	Unmyelinated	Deep and diffuse pain, temperature, postganglionic autonomic

(Snell, 1997:101; Darby and Daley, 1995:252; Jacobs and Lowe, 1999:79;)

[12] Axon is the central core of the nerve fibre, the membrane of the axon is the conductive membrane for the nerve impulse (Crossman and Neary, 1995:15).

2) Classification of nerve fibres according to their origin, function and conduction velocity.

Table 2.4. Sensory nerve classification by origin, function and velocity

Fibre type	Origin	Function/s	Conduction velocity
Type I	Mechanoreceptors	Proprioception	70 –120
Type II	Mechanoreceptors	Cutaneous information from the skin	30 –62
Type III	Mechanoreceptors	Deep pressure, touch, temperature	6 –30
Type IV	Nociceptors	Crude touch, pressure, pain and temperature	6 –16

(Darby and Daley, 1995:252; Kingsley, 1996:140; Guyton and Hall, 1997:379).

Darby and Daley, (1995:252) explained that information from mechanoreceptors are transmitted along the A- beta fibres, information from mechanical and thermal nociceptors are conducted via A-delta fibres and stimuli from polymodal nociceptors travel along C fibres. Snell (1997:121-122) added that the A- delta and C sensory nerve fibres are the afferent fibres for free nerve endings.

In analysing the two systems of classification for sensory nerve fibres it can be seen that the type IV fibres (classification according to origin, function and conduction velocity) and the type C and A- delta fibres (according to classification by nerve diameter) have similar functions in nociceptive afferents. For the purpose of this study these terms will be used interchangeably to indicate nociceptive sensory (afferent) fibres. Likewise the type I – III fibres (origin, function and conduction velocity classification) and the type A-beta fibres (nerve diameter classification) transmit mechanoreceptive information. These nerve fibres will be used interchangeably to indicate mechanoreceptive afferent fibres.

2.3.4. Transmission to the spinal cord (continued)

The spinal nerve divides into two roots near the spinal cord, the dorsal and ventral roots, which attach to the tips of the dorsal and ventral horns of the spinal cord respectively. Afferent fibres enter the spinal cord via the dorsal root to the dorsal horn. The nerve cell bodies of these neurons are located in dorsal root ganglia, which appear as small enlargements on the dorsal roots, near their convergence with the ventral roots at the entrance to the intervertebral foramina. Once passing through the intervertebral foramen the impulse enters the spinal cord (Crossman and Neary, 1995:40).

2.3.6. Ascending pathway of the spinal cord

The stimulus is conveyed via the dorsal roots of the sensory nerve to the grey matter of the spinal cord. The grey matter of the dorsal horn is divided by cytoarchitecture into 10 zones known as laminae. These laminae are numbered numerically from dorsal to ventral (Crossman and Neary, 1995:42). Sensory information terminates on the various types of laminae (I – VI), which transmit this information to higher centres (Darby and Daley, 1995:260).

The type A-delta fibres terminate on lamina I, IV, V and VI. Type C sensory fibres terminate on lamina II. Some type A-beta fibres terminate on lamina II, III, IV, V and VI (Darby and Daley, 1995:260).

Guyton and Hall (1997:385-386) added that most type A- beta fibres pass through the dorsal horn uninterrupted and terminate at the gracile and cuneate nuclei of the dorsal column in the medulla oblongata.

Laminae I – III is collectively known as the substantia gelitinosa (Crossman and Neary, 1995;42). This region receives afferent neurons that are associated with nociception. The Substantia gelatinosa therefore plays a complex role in the transmission of pain. Transmitting information to the

ascending spinothalamic and spinoreticular tract neurons (to be discussed later). The theory of pain information transmission via the substantia gelatinosa has been proposed as the gate control theory (Crossman and Neary, 1995:25).

2.3.6.1. The gate control theory

Melzack and Wall first proposed the gate control theory and described how pain was transmitted through a gate type system in the spinal cord (Cramer and Darby, 1995:364).

Sensory neurons arrive at the spinal cord via the dorsal horn. Nociception is transmitted to the spinal cord via the A- delta and C type fibres (Jacobs and Lowe, 1999:81). These fibres terminate on laminae I –III, otherwise known as the substantia gelatinosa (Cramer and Darby, 1995:364).

The substantia gelatinosa consists mainly of inhibitory interneurons that transmit general inhibitory effects to the transmission cells. Transmission cells are dorsal horn neuronal projections that relay C fibre activity to higher centres via the spinothalamic tract of the anterolateral system (Cramer and Darby, 1995:364 and Wood, 1998:85).

Predominance in C fibre activity “opens” the “gate” to pain transmission to higher centres. The gate is opened by C fibre activity, which inhibits the effects of the substantia gelatinosa on the transmission cells, allowing the transmission of pain (Jacobs and Lowe, 1999:82 and Wood, 1998:85).

Conversely, a predominance of A-Beta fibres closes the gate. These fibres facilitate the inhibitory effect of the substantia gelatinosa. Therefore closing the gate and stopping pain

> transmission to the transmission cells (Jacobs and Lowe, 1999:82 and Wood, 1998:85).

Information in the laminae ascends via the spinothalamic and spinorecticular tracts to the recticular nuclei in the brain stem (Jacobs and Lowe, 1999:83-87).

> The spinothalamic tract conducts information related to pain, thermal sensations, non-discriminate touch and pressure (Guyton and Hall, 1997:386).
>
> The spinothalamic neurons decussate to the contra-lateral spinothalamic tract, usually within one spinal segment (Guyton and Hall, 1997:386).
>
> The spinoreticular tract represents a route by which sensory impulses ascend to higher centres. Information is conveyed from the dorsal horn of the spinal cord to the brainstem reticular formation. This route is thought to be responsible for the transfer of dull, aching pain (Crossman and Neary, 1995:50).

From the ascending tracts the impulse is carried via the medial lemniscus to the ventropost-lateral nucleus of the thalamus and further to the cerebral cortex (Hopkins and Ingersoll, 2000).

2.3.7. Role of the cortex in muscle response

Cervero *et al* (1991) proposed that joint injury causes a disruption in the afferent activity (sensory input) from the joint, which results in inhibition of the joint's motor neurons due to a reflex arc mechanism that is mediated by supraspinal structures (Guyton and Hall, 1997:442). The reflex arc causes a decrease in the inhibition of the inhibitory mechanism, allowing inhibition of the motor neurons and the presence of arthrogenic muscle inhibition (Hopkins and Ingersoll, 2000).

This explanation was supported by Valeriani *et al.* (1996), who explained how the functioning of the central somatosensory pathways are modified by lesions to peripheral mechanoreceptors. However, Werner *et al.* (1991) suggested that joint afferents do not change cortex activity. They expanded on this suggestion by stating that stimulation of afferent neurons showed primary cortex activity that directly correlated to the EMG of the muscle.

Although it has been suggested that the cortex is involved in the complex integration of articular inputs from proprioceptors, it has been shown that joint afferents could influence the cortex response (Hopkins and Ingersoll, 2000).

2.3.8. Descending pathways in the spinal cord

The information from supraspinal centres is transmitted via descending pathways. The information is conveyed along specific relevant spinal tracts (Hopkins and Ingersoll, 2000).

The descending spinal tracts include the corticospinal, vestibulospinal and rubrospinal tracts.

2.3.8.1. Corticospinal tract

This tract consists of approximately one million axons. The function of the tract is to conduct motor information from the motor and parietal cortices to motorneurons. The tract plays a role in governing the force of muscle contraction that is generated (Porter and Wilkinson, 1997:248). Hence, the corticospinal tract transmits important information regarding voluntary muscle contraction (Darby and Daley, 1995:276). The neurons from this tract synapse on interneurons, and alpha and gamma motorneurons (Hopkins and Ingersoll, 2000).

The tract has mostly a facilitatory function, although some inhibitory responses have been recorded. Inhibition may be caused by neurons from the tract converging on inhibitory interneurons, the response from which would be an inhibition of the normal afferent activity and subsequent motor response. The role that the corticospinal system plays in arthrogenic muscle inhibition is not completely understood (Hopkins and Ingersoll, 2000).

2.3.8.2. Vestibulospinal tract

This tract is activated in postural reflexes. The tract transmits information from the lateral vestibular nucleus of the fourth ventricle to the motor neurons and interneurons (Darby and Daley, 1995:280). The information that is conveyed via this tract is crucial in maintaining upright posture. This tract is active during the initiation of voluntary movement to cause reflex postural changes, which are mediated by interneurons and the cerebral cortex (Hopkins and Ingersoll, 2000).

2.3.8.3. Rubrospinal tract

This tract consists of a smaller amount of neurons than compared with the other descending tracts. The tract originates in the red nucleus of the midbrain and terminates on distal motor neurons. It is involved in the innervation of musculature and has been identified as a source of inhibition for interneurons (Hopkins and Ingersoll, 2000).

The descending tracts finally terminate principally on the interneurons in the intermediate regions of the cord gray matter and there in turn excite the anterior motor neurons that cause muscle contraction (Guyton and Hall, 1997:455).

2.3.9. Interneurons

Ingersoll, Palmieri and Hopkins (2003) proposed that interneuron activity was responsible for the development of arthrogenic muscle inhibition.

An interneuron is a nerve fibre that receives and transmits information from one nerve fibre to another; they are the intermediaries in neural pathways (Hopkins and Ingersoll, 2000).

Interneurons receive input from:

- Sensory afferent fibres.
- Descending fibres.
- Other interneurons.

Interneurons relay information on to:

- Alpha[13] and gamma[14] motor neuron pools.
- Autonomic efferent neurons.

[13] Alpha motor neurons innervate large skeletal muscle fibres through large A-alpha nerve fibres (Guyton and Hall, 1997:441).
[14] Gamma motor neurons cause contraction of skeletal muscles, signals are transmitted via the A-gamma fibres (Guyton and Hall, 1997:441).

- Ascending pathways.

(Hopkins and Ingersoll, 2000).

Initially it may appear as though interneurons function as relay stations, they do however, play an important integrative function (Hopkins and Ingersoll, (2000). Hopkins and Ingersoll, (2000) described how the axons of interneurons ascend or descend in the white matter of the spinal cord for two or three levels before entering the gray matter.

Hopkins and Ingersoll (2000) classified interneurons into type Ia inhibitory interneurons and type Ib inhibitory or excitatory interneurons. The Ia inhibitory interneurons have been found to be active during reciprocal inhibition (Hopkins and Ingersoll, 2000).

Reciprocal inhibition is caused by the stimulation of muscle spindle afferents. The result is a direct excitation of the affected motorneuron pool and a reflex stimulation of the Ia inhibitory interneuron, which causes the inhibition of the antagonist muscle. This allows for the smooth co-ordination of voluntary muscle contraction without the interference from the antagonist muscles (Ingersol, Palmieri and Hopkins, 2003).

The complexity of the interneural system is further complicated by the identification by Jacobs and Lowe (1999:81) that the interneurons receive information from the corticospinal, rubrospinal, vestibulospinal tracts and renshaw cells. Interneurons thus receive and integrate information from supraspinal structures. The net effect of interneurons is either inhibition or excitation of the motorneuron pool, which is dependent on the integration of information from all these systems (Ingersoll, Palmieri and Hopkins, 2003).

Hopkins and Ingersoll (2000) proposed that the renshaw cells cause inhibition of the Inhibitory Ia interneurons, this inhibition is known as disinhibition or excitation of the interneuron.

The presence of Ib excitatory and inhibitory interneurons have been identified (Hopkins and Ingersoll, 2000), with these interneurons receiving information from:

- Golgi tendon organs
- Joint and cutaneous efferents[15]
- Inhibitory Ia interneurons
- Descending tracts from the brain stem

Injury to a joint results in the activation of joint receptors that appear to stimulate the type Ib inhibitory interneurons (Hopkins and Ingersoll, 2000). In turn the Ib inhibitory interneurons have been found to inhibit the A-alpha motor neurons that are responsible for the force of contraction of the innervated muscle, this results in decreased strength of the muscle contraction (Iyer, Mitz, and Winstein, 1999:230).

Therefore, Ingersoll, Palmieri and Hopkins (2003) proposed that interneuron activity was responsible for the development of arthrogenic muscle inhibition. The effect of all information arriving at the interneuron is expressed in either an inhibitory or excitatory response of the motor neuron pool.

Normally, neurons from descending tracts converge on inhibitory interneurons, causing inhibition of the inhibitory mechanism, therefore resulting in excitation (Hopkins and Ingersoll, 2000). This normal situation would stop the development of arthrogenic muscle inhibition and the inhibition of the related joint musculature (Hopkins and Ingersoll, 2000).

Interneurons excite the anterior motor (efferent) neurons that are responsible for muscle contraction (Guyton and Hall, 1997:455).

[15] The motor or other effector function of a neural element (Redwood, 1997:336).

2.3.10. Motor Neurons and the Motor Neuron Pool

Motor neurons

According to classification by diameter there are two different types of motor neurons; namely the alpha, and gamma motor neurons. These are efferent fibres, meaning that they are the effector neurons of a neural element (Redwood, 1997:336).

Table 2.5. Efferent nerve classification

Fibre type	Fibre Diameter	Myelination	Functions
Alpha (α)	12 - 20	Heavily myelinated	Motor
Gamma (γ)	3 - 6	Myelinated	Motor to muscle spindle

(Snell, 1997:101; Darby and Daley, 1995:252; Jacobs and Lowe, 1999:79;)

The function of motor neurons is to innervate skeletal muscle and regulate the contraction of these muscles (Darby and Daley, 1995:283).

Alpha motor neurons exclusively innervate the extrafusal fibres in skeletal muscle. These neurons are the largest and fastest of the motor neurons (Iyer, Mitz, and Winstein, 1999:220).

Conversely, the gamma motor neurons innervate the intrafusal fibres of the muscle spindle, and are the smallest and slowest motor neurons (Iyer, Mitz and Winstein, 1999:220).

Motor neuron pool

Both types of motor neurons combine together to form a motor neuron pool, which is responsible for the innervation of one particular muscle group. The motor neuron pool and the muscle fibres they innervate work as a unit, which is commonly known as a motor unit (Darby and Daley, 1995:284). The number of motor units that are recruited in a muscle contraction governs the strength of the contraction i.e. the more motor units that are recruited the greater the muscle contraction (Iyer, Mitz and Winstein, 1999:221).

2.4. ARTHROGENIC MUSCLE INHIBITION

2.4.1. Definition

Arthrogenic muscle inhibition is a presynaptic, ongoing reflex inhibition of musculature surrounding a joint. It is a natural response following distension or damage to structures in the joint (Hopkins and Ingersoll, 2000).

2.4.2. Introduction

Arthrogenic muscle inhibition is the inability of a functional muscle group to recruit all their motor units during a maximal voluntary effort (Suter, *et al.* 2000). Clinically arthrogenic muscle inhibition manifests itself as a decrease in strength of the affected muscle group (Suter, *et al.* 1999).

Most studies on arthrogenic muscle inhibition have focussed on the knee joint and its' effects on the quadriceps muscle, little emphasis has been placed on the hip (Arokoski, *et al.* 2002).

2.4.3. The cause of Arthrogenic muscle inhibition

Arthrogenic muscle inhibition is caused by activity from many different joint receptors, which act on inhibitory interneurons synapsing on the motorneuron pool of joint musculature. The information from inhibitory .interneurons impedes the recruitment within the motorneuron pool, decreasing the force of any contraction originating from that motorneuron pool. Free nerve endings and specialised nociceptors may play a role in inhibition, but the primary effect seems to be as a result of mechanoreceptor activity (Ingersoll, Palmieri and Hopkins, 2003).

The joint receptors are stimulated by pain, ligament stretching, capsule compression, effusion or irritation due to injury within the joint (Spencer, Hayes and Alexander, 1984).

Arokoski, *et al.* (2002) proposed that the weakness seen in muscles moving joints that are affected by osteoarthritis was partly caused by reflex inhibition from the joint, due to stimulation of the joint receptors.

2.4.4. The clinical effect

Arthrogenic muscle inhibition is clinically important because it causes a decrease in strength of the affected muscle group (Hurley, Jones and Newham, 1994). Suter *et al.* (2000) stated that arthrogenic muscle inhibition may limit the functional recovery of the joint and muscle complex after injury, and that the early goals of treatment should be to reduce muscle inhibition to gain full recovery.

However, Ingersoll, Palmieri and Hopkins (2003) believed that arthrogenic muscle inhibition is a natural response of the joint in order to protect itself. They believed removing the inhibition to soon might make the patient prone to reinjuries due to the patient's enhanced ability to move the injured joint. Ingersoll, Palmieri and Hopkins (2003) proposed that in order to maximise the benefits of reducing arthrogenic muscle inhibition while minimising the risks of reinjury, the patient should be treated and supervised in a controlled environment.

After joint injury has caused arthrogenic muscle inhibition and subsequent weakness to set in, the evident decrease in strength hampers the rehabilitation process of the injured joint despite complete muscle integrity (Suter, *et al.* 2000). Exercise is important to increase healing and prevent a multifaceted injury paradigm (Hopkins and Ingersoll, 2000).

The removal of arthrogenic muscle inhibition allows patient's to maintain or increase activity levels causing a decrease in rehabilitation time, a quicker

return to activity and a reduction of the adverse effects of arthrogenic muscle inhibition on tissues. The prolonged presence of arthrogenic muscle inhibition can cause damage to muscles, bones, ligaments and nerves (Hopkins and Ingersoll, 2000).

Arthrogenic muscle inhibition can lead to atrophy of the muscles surrounding the affected joint, which will further hamper the rehabilitation process (Hurley, Jones and Newham, 1994).

2.4.5. Natural progression

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 (Nordin and Frankel, 1989 as cited by Ingersoll, Palmieri and Hopkins, 2003).

In muscle, we may see type I fibre atrophy, decreased cross-sectional area and decreased oxidative enzyme activity (Hurley, Jones and Newham, 1994).

The effects on bone include periosteal and sub-periosteal resorption, decreased strength, diminished load to failure and a lower energy storage capacity (White and Panjabi, 1990:264).

Ligaments have been found to become elongated and less stiff, with a decreased load to failure capacity and a reduction in tensile strength (White and Panjabi, 1990:22).

Negative neural factors include depolarised muscle fibre membrane, decreased potential across the motor-end plates and reduced potassium and sodium transport across the membranes (Ingersoll, Palmieri and Hopkins, 2003).

In spite of these adverse effects of arthrogenic muscle inhibition, the removal of AMI is not generally stated as a goal of rehabilitation. Arthrogenic muscle inhibition is not given the attention it deserves because it is often seen as being unalterable or able to resolve itself given time, which is often not the case (Ingersoll, Palmieri and Hopkins, 2003).

2.4.6. Arthrogenic muscle inhibition in the contra-lateral limb

Young (1993) described how arthrogenic muscle inhibition can be present in the contra-lateral limb. Suter *et al.* (1998) supported this statement by showing how the non-involved limb showed the presence of arthrogenic muscle inhibition that was at a level higher than a healthy population.

The explanation for the effects of arthrogenic muscle inhibition being present in the opposite lower limb can be two fold:

1. Injury of a joint may lead to the alteration of the normal muscle activity and normal gait cycle. This would therefore, cause a disruption of the neuromuscular control of the involved muscles (Suter, *et al.* 1998).

2. Neural pathways have connections in the spinal cord which could cause transfer of inflammation towards the contra-lateral side (Suter, *et al.*1998).

Hopkins and Ingersoll (2000) lend support to the second explanation offered by Suter, *et al.* (1998) by identifying how crossed spinal pathways transmit information to the contra-lateral leg, which could transfer the neurological information of arthrogenic muscle inhibition to thye contra-lateral side.

The presence of arthrogenic muscle inhibition in the contra-lateral leg has eliminated the possibility of the opposite leg as a control in experimental investigations and measurements (Suter, *et al.* 1998).

2.4.7. Measurement of arthrogenic muscle inhibition

Arthrogenic muscle inhibition causes a reduction in motor neuron recruitment, which can clinically be seen as a decrease in the force of contraction of the affected muscle (Suter, *et al.* 2000). Measurement of the presence of inhibition can be achieved in various ways.

> Voluntary measurements can be taken using a dynamometer or electromyography. These measurements will give an indication into the force of contraction used by the muscle, which is an indirect evaluation of the muscles' motor neuron recruitment (Perrin, 1993:213).
>
> Involuntary measurements of motorneuron recruitment can be made through careful stimulation of sensory fibres and the evaluation of the reflexive twitch contraction using the Hoffmann reflex (Hopkins and Ingersoll, 2000).
>
> Other means of measurements include interpolated twitch techniques, this combines voluntary muscle contraction with a superimposed electrical impulse (Ingersoll, Palmieri and Hopkins, 2003).

Each method has their advantages and disadvantages, which will be discussed in the following paragraphs.

2.4.7.1. Voluntary force measurement

A decrease in the force of muscle contraction is one of the final outcomes of arthrogenic muscle inhibition. The difference between a baseline maximum voluntary force contraction (MVC) before joint injury compared to a MVC after joint injury will give an

indication into the extent and presence of inhibition (Hopkins and Ingersoll, 2000).

The therapeutic use of voluntary force measurements is limited when trying to establish the presence of inhibition, unless a baseline MVC reading was taken prior to the joint injury. The patient's perception of pain and lack of confidence in maximally moving a previously injured joint further limits the force measurements. The patient has to be able and willing to perform the contraction at the patients' maximum for the results to be of use (Ingersoll, Palmieri and Hopkins, 2003).

Stokes and Young (1984) compared force measurements of the injured leg to the contra-lateral (or uninjured) leg to measure inhibition in the injured leg. Hopkins and Ingersoll (2000) refuted the validity of this comparison by explaining that crossed spinal pathways may transmit information to the uninvolved leg, which may cause inhibition of the joint musculature of the contra-lateral leg.

During force measurement synergistic muscles that may not be inhibited will contribute to the action being tested, voluntary force measurements are unable to test one muscle independently. This questions the accuracy of voluntary force muscle testing (Ingersoll, Palmieri and Hopkins, 2003).

Interpolated twitch technique is a combination of a MVC and a supramaximal external stimulus. The stimulus is added to compensate for the inhibited portion of the motor neuron pool. This method allows for the measurement of inhibition but without the need for a baseline torque measurement. The force of contraction resulting from the twitch response is very small compared with the potential force of the muscle, thus the twitch may go undetected (Hopkins and Ingersoll, 2000).

2.4.7.2. Involuntary measurement (The Hoffmann reflex)

The Hoffmann reflex (H-reflex) is an indirect means of measuring motor neuron pool recruitment. The H-reflex is initiated by the stimulation of a mixed nerve, which causes a twitch response of the innervated muscle. Assessing the grade of the twitch response indicates the amount of motor neuron pool recruitment in the muscle. The muscle activity is visualised by surface electromyography (Ingersoll, Palmieri, and Hopkins, 2003).

The stimulus is applied to a mixed nerve, which results in an electromyograhic response. The electromyographic response is caused by the development of an action potential along the type I alpha nerve fibres to the alpha motor neuron pool in the anterior horn of the spinal cord. This neural pathway constitutes the H-reflex (Hopkins and Ingersoll, 2000).

The larger the intensity of the stimulus the larger the number of afferent fibres that are stimulated, and in turn, more motor neurons are recruited within the motor neuron pool. This results in larger amplitude of the twitch response in the affected muscle. The amplitude of the twitch response reflects the portion of the motor neuron pool that was stimulated by the afferent neural activity. Therefore inhibition will decrease the excitability of the motor neuron pool, with a subsequent reduction in the amplitude of the H-reflex (Hopkins and Ingersoll, 2000).

Assessing the H-reflex allows for a single muscle to be tested, independent of its synergistic muscles. The H-reflex is performed whilst the subject is resting because no voluntary contraction is required. This allows for assessment in pathological subjects where voluntary contraction of the affected muscle may have been restricted. The H-reflex has the added benefit of being a very sensitive assessment tool, it is able to detect small changes

in the motor neuron poll excitability (Hopkins and Ingersoll, 2000). The H-reflex however, does not take into consideration supraspinal inputs that may affect the motor neuron pool during voluntary contraction (Ingersoll, Palmieri and Hopkins, 2003).

Isokinetic muscle testing, a form of voluntary muscle force measurement was chosen as the objective measurement tool for this study and will be discussed in further detail at the end of this chapter.

2.4.8. Treatment of Arthrogenic muscle inhibition

Investigations into the treatment of arthrogenic muscle inhibition have shown limited success with various modalities; these modalities and their efficacy are discussed below.

Treatment of arthrogenic muscle inhibition is aimed at removing, masking, or overriding the inhibitory interneuron activity. Current options in overriding the interneuron inhibition are aimed at stimulating the efferent fibers of the sensory component of the peripheral nervous system (Ingersoll, Palmieri, Hopkins, 2003).

Possible treatments that might be beneficial in reducing arthrogenic muscle inhibition include lidocaine injection into the joint, cryotherapy and TENS (Transcutaneous electrical nerve stimulation) (Hopkins and Ingersoll, 2000).

Cryotherapy causes reduction in nerve conduction velocity, synaptic transmission, muscle spasm and pain. All these effects would be beneficial in the treatment of arthrogenic muscle inhibition. However the most significant effect of cryotherapy in the treatment of arthrogenic muscle inhibition is the definite effect of slowing and eventual blocking of the sensory nerve fibres. The effects seem to be linear where the cooler the nerve is, the slower the impulse is carried (Knight, 1995).

TENS causes stimulation of the cutaneous type I nerve endings, which could compete for the same type I afferent fibers that carry information from joint receptors to the spinal cord. This competition is believed to block the signal of inhibition from joint receptors, thus reducing arthrogenic muscle inhibition (Ingersoll, Palmieri and Hopkins, 2003).

Hopkins *et al.* (2001) suggested that both cryotherapy and TENS caused disinhibition of the quadriceps motorneuron pool following knee joint effusion. The effects of cryotherapy where present during cooling and during the 30-minute post-cooling phase. The effects of TENS on reducing arthrogenic muscle inhibition was found to be present only whilst the modality was applied, there was no lasting effect once the modality was removed.

Lidocaine injection was shown to bring temporary relief of the symptoms of arthrogenic muscle inihibition (Ingersoll, Palmieri and Hopkins, 2003).

The success of conservative treatment in restoring muscle function is limited in the presence of serve muscle inhibition (Suter, *et al.* 1999).

Suter *et al.* (1999) investigated the effects of sacroiliac manipulation on quadriceps strength in patients suffering from anterior knee pain. In their study they noted a significant increase in quadriceps strength after the manipulation. In a follow-up study (Suter *et al.* 2000) using a randomised, double blinded, controlled clinical trial the same results were produced, however the inverse relationship of muscle strength and muscle inhibition did not reach a statistically significant level. The results of both of their trials could have been compromised from the inclusion of subjects who had previous knee surgery. Inclusion of these subjects would have disrupted the homogeneity of their sample.

In an explanation of their results Suter *et al.* (1999 and 2000) proposed that the manipulation caused activation of mechanoreceptors and proprioceptors from structures in and around the joint. This caused an

altered afferent input into the motorneuron pool resulting in a disruption of the pain-spasm–pain cycle. The proposed altered afferent input caused a decrease in muscle inhibition demonstrated by the increase in strength. Suter *et al.* (1999) was able to speculate on chiropractic treatment forming an alternative or adjunct to traditional rehabilitation in patients with anterior knee pain and muscle inhibition.

2.4.9. The proposed mechanism of chiropractic manipulation in the treatment of arthrogenic muscle inhibition

When articular surfaces are separated during manipulation it causes stretching of the musculature adjacent to the joint. Muscles surrounding a dysfunctional joint are often hypertonic. A manipulation causes the elongation of muscle spindles in these hypertonic muscles. Reflexes mediated by the muscle spindles relieve this hypertonicity (Leach, 1994:44). Likewise, manipulation causes stretching of the joint capsule and stimulation of mechanoreceptors, which causes the reflex inhibition of facilitated motorneuron pools that are responsible for the increased muscle tone and spasm (Kirkaldy-Willis and Burton, 1992:288; Colloca, 1997:47).

Following manipulation, presynaptic nociceptive inhibition of proprioceptors occurs (Lopes, 1992:66). Manipulation has been hypothesised to produce significant short-term bursts of proprioceptive transmission in Type A-alpha afferent fibres from the joint capsules, ligaments and in the muscle spindles of the local musculature. These larger fibre signals are believed to modulate the interneuronal pool via the dorsal spinal root ganglion and the substantia gelatinosa subsequently closing the gate on pain transmission (Dhami and DeBoer, 1992:121). Stimulation of nociceptors can cause complex reflexes, which effect the sympathetic nervous system, causing an increase in muscle tone of the area. Therefore, if manipulation causes an interruption or decrease in nociceptive input to the central nervous system it would inhibit the complex sympathetic mechanisms that cause the initial muscle spasm, helping to restore the muscle back to its normal state and to the muscles normal levels of contraction (Colloca, 1997).

Reduction of the spasm surrounding the joint can be mediated by the stimulation of mechanoreceptors within the adjusted joint causing reflex neural mechanisms that restore the inhibition of the normal inhibitory mechanism (Ingersoll, Palmieri and Hopkins, 2003).

2.4.10. Arthrogenic muscle inhibition at the sacroiliac joint

Sakamoto, *et al.* (2001) reported that the sacroiliac joint was richly innervated with nociceptors. The authors conducted a histological examination on feline subjects, where they identified mechanosensitive afferent units that innervated the joint and adjacent tissue. Sakamoto *et al.* (2001) concluded that of the 29 units they were able to identify, 26 were presumed to be nociceptors and the remaining 3 fibres were thought to have a proprioceptive function.

Vilensky *et al.* (2002) was able to support the presence of mechanoreceptors in the human sacroiliac joint. Using histology and immunohistochemical techniques they were able to identify paciniform nerve endings, non-paciniform nerve endings and free nerve endings in the posterior ligament of the human sacroiliac joint.

Therefore the sacroiliac joint and peri-articular structures contain numerous mechanoreceptors and nociceptors (Fortin *et al.* 1999). It can be presumed that the sacroiliac joint transmits information on nociception and proprioception to the central nervous system (Hopkins and Ingersoll, 2000), due to the substantial innervation of the sacroiliac joint with mechanoreceptors (Sakamoto *et al.*, 2001). This in turn may result in the development of arthrogenic muscle inhibition in the muscles that fall within the motor neuron pool of the affected sacroiliac joint.

2.4.11. The sacroiliac joint as a source of low back pain

In the search for the cause of low back pain the sacroiliac joint has gained interest as a pain generator (Indahl, *et al.* 1999). These findings are in keeping with Schwarzer, Aprill and Bogduk (1995), who found that the sacroiliac joint is a significant source of pain in patients who suffer from chronic low back pain and warrants further study.

Sakamoto, *et al.* (2001) reported that the sacroiliac joint is responsible for 22.6% of low back pain cases, which concurs with medical literature that has estimated the prevalence of sacroiliac dysfunction in the population to be between 19.3% and 47.9% (Toussaint, *et al.* 1999).

Loosening, due to any cause, of the self-tightening mechanism of the sacroiliac joint will lead to static insufficiency (Hendler *et al.* 1995).

The sacroiliac joint is a significant source of low back pain (Murata, *et al.*, 2001). The predominance in nociception of this joint will lead to the opening of the “gate” to pain, as described earlier in the gate control theory of pain (Jacobs and Lowe, 1999:82). This in turn may lead to the development of arthrogenic muscle inhibition in the muscles that fall within the motor neuron pool of the affected sacroiliac joint.

2.5. SACROILIAC JOINT SYNDROME

2.5.1. Causative factors

Hendler, *et al.* (1995) stated that the sacroiliac joint syndrome is attributable to a sacroiliac joint subluxation. The subluxation was caused by the slipping of the ilium on the sacrum, resulting in the ridge of one articular surface being wedged on the ridge of the other articular surface. This results in tightening of the ligaments, a reflex muscle spasm of the surrounding musculature and intense pain.

2.5.2. Presentation

The patient's pain is often not relieved by sitting or recumbency. In the sitting position the patient appears most comfortable sitting on the unaffected buttock and in the forward flexed position (Hendler, *et al.* 1995).

2.5.3. Symptoms

Patients present with pain over the sacroiliac region, which is often localised over the posterior superior iliac spine. Referred pain can be felt over the buttock, groin, and leg (Kirkaldy-Willis and Burton, 1992:123).

Hendler, *et al.* (1995) added that the referred pain is often felt in the groin, posterior thigh and less commonly on the lateral calf.

These symptoms are often exacerbated by daily living, especially climbing stairs (Daum, 1995).

2.5.4. Clinical signs

The symptomatic sacroiliac joint is usually tender to palpation, with evidence of asymmetry of movement within the joint (McCulloch and Transfeldt, 1997:180-181).

Provocation tests like Gaenslen's and Patrick Faber reproduce or exacerbate the pain in the joint. These tests help to confirm the diagnosis (McCulloch and Transfeldt, 1997:180-181).

2.5.5. Physical testing

Physical testing involves the use of provocation tests to stress the joint's structure in an attempt to reproduce the patient's pain (Laslett and Williams, 1994).

The following provocation tests were used in this to aid in the diagnosis of a sacroiliac syndrome:

Posterior shear (Kirkaldy-Willis and Burton, 1992:125)
Gaenslen's test
Patrick FABER
Yeoman's test
(Magee, 2002:588-620)

Laslett and Williams (1994) found that when using provocation tests for diagnostic purposes, only those tests that have a proven, high inter-examiner reliability should be used. This will ensure the validity of the diagnosis. In their study to assess inter-reliability of seven pain provocation tests for pain of sacroiliac origin they found that Gaenslen's test and the posterior shear test had the greatest inter-examiner reliability of the tests they tested.

A double-blinded study, conducted by Broadhurst and Bond (1998), investigated the specificity and sensitivity of the Posterior shear and

Patrick Faber tests in detecting sacroiliac joint dysfunction. They were able to conclude that both tests had a high level of sensitivity (77%) and specificity (100%) for the detection of sacroiliac dysfunction.

However, Yeoman's test was found to be the most reliable and specific test for the diagnosis of sacroiliac syndrome in a study conducted by Kirkaldy-Willis and Burton (1992).

Riggien (2003) in assessing the reliability and validity of the orthopaedic rating scale found that the reliability and validity of tests for sacroiliac syndrome were low and recommended that a combination of equally scoring tests be applied in order for consistent diagnosis of sacroiliac syndrome.

The reliability and specificity of provocation tests for sacroiliac dysfunction are questionable, as research has produced inconsistent results (Vleeming, *et al.* 1990).

To compensate for this dispute Cibulka and Koldehoff (1999) suggested gaining at least three positive provocation test results in order to validate a diagnosis of sacroiliac dysfunction.

2.5.6. Treatment

In a literature review on the sacroiliac joints' role in generating pain, Daum (1995) outlined various options in the treatment of sacroiliac dysfunction. He proposed that a symptomatic sacroiliac dysfunction is frequently a self-limiting condition that responds well to conservative treatment.

Conservative treatment for this condition should include the limitation of activities that exacerbate the pain and increase the forces within the sacroiliac joint (Daum, 1995). Restricted activities should include excessive bending and lifting. Education should be given on proper lifting techniques and the correct procedure for getting out of bed (Hendler *et al.* 1995).

Harrison, Harrison and Troyanovich (1997) mentioned the importance of performing rehabilitation exercises that will enhance the function of the self-bracing system, which will help prevent future recurrences of the condition. Sacroiliac belts can also be used, these provide added bracing to the joint (Daum, 1995).

Daum (1995) proposed that the aim of physical therapy should be to reduce and stabilise the instability or subluxation. This can be achieved by a series of mobilisations and manipulations combined with pelvic strengthening exercises. If this treatment fails after six weeks, injections of anaesthetics and steroids can be administered. Non-steroidal anti-inflammatory agents can be given, provided the patient shows no contraindications to its use.

The use of manipulation is useful in the reduction of sacroiliac dysfunction (Hendler, *et al.* 1995). Daily manipulation for up to 10 days was reported by Hendler *et al.* (1995) to have success rates as high as 90%. Further to this Herzog, *et al.* (1991) compared the treatment outcomes between a back school programme and spinal manipulative therapy on patients suffering from sacroiliac dysfunction. From their comparison they concluded that spinal manipulative therapy was more effective in restoring normal gait symmetry (objective measures) than a back school programme. However, they (Herzog, *et al.* 1991) also concluded that the back school programme was more effective than spinal manipulation when comparing clinical outcomes (subjective measures).

Chiropractic care has been found by many studies, literature reviews and opinions to be more cost effective, safer and appropriate than other treatment options like: drug therapy, bed rest, physical therapy and surgery (Cooperstein, *et al.* 2001).

2.5.7. Chiropractic Care for sacroiliac syndrome

The effectiveness of manipulation in the treatment of sacroiliac syndrome has a large support base (Cooperstein, *et al.* 2001).

Manipulation is thought to restore joint play to dysfunctional joints. Shakelle (1994), proposed that manipulation restores normal joint play by causing:

- The release of entrapped synovial folds or plica,
- The relaxation of hypertonic muscles,
- The disruption of articular and peri-articular adhesions.

Some of the effects of manipulation were outlined by Calliet (1981) as follows:

- Manipulation causes the abrupt movement of the joint, which leads to the desensitisation of the mechanoreceptors and removal of the reflex muscle spasm. This allows the joint to move more freely.
- Manipulation allows the capsule to be freed
- The dynamic thrust[16] of the manipulation causes stimulation of the muscle spindles in the adjacent musculature. This leads to reciprocal relaxation of the extrafusal muscle fibres. This helps to reduce the muscle spasm found around the joint.

[16] A brief sudden, and carefully administered impulsion that is given at the end of the range of the normal passive range of movement. It is usually accompanied by a cracking noise. (Kirkaldy-Willis and Burton, 1992:283).

- The malaligned spinal segments are aligned to conform to the centre of gravity.

Indahl, *et al.* (1999) speculated that stimulation of receptors in the joint capsule lead to reflex muscle spasm. Defranca (1996:295) proposed that stimulation of joint mechanoreceptors during manipulation creates reflex changes in the tone of the muscles that serve the joint.

Hopkins, *et al.* (2000); Suter, *et al.* (1999) and Suter *et al.* (2000) supported the proposal made by Defranca (1996) and added that manipulation plays a role in the reduction of arthrogenic muscle inhibition. After manipulation of the affected joint an increase in strength of the joint's musculature was observed.

Due to the supporting literature the use of manipulation is indicated for the treatment of sacroiliac joint syndrome.

2.5.8. Conclusion

Arthrogenic muscle inhibition is caused by the stimulation of joint mechanoreceptors (Suter, *et al.* 2000). The sacroiliac joint is richly innervated with nociceptive mechanoreceptors (Sakamoto, *et al.* 2001). A sacroiliac syndrome will cause stimulation of these nociceptors due to the perceived pain (Cooperstein, *et al.* 2001). It is of the authors' opinion that stimulation of these nociceptors as in the case of a symptomatic sacroiliac syndrome, may lead to the development of arthrogenic muscle inhibition originating in the sacroiliac joint. Arthrogenic muscle inhibition will cause the inhibition of the muscles that are within the motorneuron pool of the affected joint (Hopkins and Ingersoll, 2000). Therefore, arthrogenic muscle inhibition originating in the sacroiliac joint will cause the inhibition of muscles that fall within the motor neuron pool of the sacroiliac joint.

The actions responsible for movement at the hip fall into the motorneuron pool of the sacroiliac joint (Moore and Dalley, 1999: 540).

Thus a symptomatic sacroiliac syndrome could lead, ultimately, to the inhibition of the actions of the hip, through the development of arthrogenic muscle inhibition.

2.6. HIP JOINT

2.6.1. The neurological link between the sacroiliac joint and the hip musculature

Murata, *et al.* (2001) investigated the sensory innervation of the sacroiliac joint in rats. Rats and humans have a similar relationship between the sacroiliac joint and the lumbosacral plexus, despite the presence of a sixth lumbar vertebra in rats. From their experiment they (Murata, *et al.* 2001) were able to conclude that the sacroiliac joint was innervated by sensory neurons in dorsal root ganglions from level L1 to S2 on the same side of the joint. Suter, *et al.* (1999) and Suter, *et al.* (2000) concurred with Murata, *et al.* (2001) on the innervation of the sacroiliac joint. They (Suter *et al.* 1999 and 2000) described the sacroiliac joint as being innervated by the anterior primary rami of L2 to S2 spinal segments.

The musculature of the hip joint receives motor innervation from segmental nerve supply of L1 to S2 (Moore 1992:476). Their individual actions and segmental nerve supply are described in table 2.6.8. The muscles responsible for movement at the hip fall into the motor neuron pool of the sensory innervation of the sacroiliac joint. Arthrogenic muscle inhibition originating at the sacroiliac joint could be responsible for inhibition of the hip musculature.

2.6.2. Anatomy of the hip joint

The hip joint is formed by the articulation of the head of the femur with the acetabulum of the hip bone. This articulation is a multi-axial ball and socket type synovial joint (Moore and Dalley, 1999:607).

2.6.3. Articular surfaces

The femoral head is sphere shaped, of which two-thirds are covered by hyaline cartilage and forms the articular surface of the hip joint. More than half of the femoral head is contained within the acetabulum. The wide superior part forms the area for weight bearing (Moore and Dalley, 1999:607).

The articular surface of the acetabulum is horseshoe shaped. A centrally located non-articular fossa is located within the acetabulum. This fossa contains a fatpad that is thin and transparent (Moore, 1992:472).

2.6.4. Acetabular labrum

The acetabular labrum is made of fibrocartilage and functions to increase the depth of the joint to accommodate the femoral head. It is attached to the bony rim of the acetabulum and adds stability to the joint (Moore, 1992:473).

2.6.5. Joint capsule

A strong dense fibrous capsule surrounds the joint at the edge of the acetabulum to the neck of the femur. The attachments to the neck of the femur are anteriorly at the intertrochanteric line and posteriorly to the intertrochanteric crest (Moore and Dalley, 1999:611).

The arrangement of the fibres in the joint capsule strengthens the joint and helps to hold the femoral head in the acetabulum (Moore, 1992:472).

2.6.6. Ligaments of the hip joint

2.6.6.1. iliofemoral ligament (Moore and Dalley, 1999:607).

This strong, Y-shaped ligament is found at the anterior aspect of the joint. It is attaches the inferior iliac spine and acetabular ridge to the intertrochanteric line of the femur.

This ligament is tense on full extension of the hip. It functions to prevent over extension whilst in the standing position and plays a role in maintaining the integrity of the joint.

2.6.6.2. Pubofemoral ligament (Moore and Dalley, 1999:611).

The pubofemoral ligament originates from the pubic part of the acetabulum and illiopubic eminence and blends in with the medial aspect of the illiofemoral ligament.

This ligament strengthens the inferior and anterior aspects of the capsule. It is taut on extension and thigh abduction. It plays an important role in preventing over abduction of the thigh.

2.6.6.3. Ischiofemoral ligament

The ischiofemoral ligament reinforces the posterior aspect of the joint by attaching proximally to the ischial portion of the acetabular rim and distally to the neck of the femur. The ligament holds the femoral head medially in the acetabulum and plays a role in preventing hyperextension of the hip (Moore and Dalley, 1999:611).

2.6.6.4. Ligament of the head of femur

This is an intra-capsular ligament. It is weak and seems to play no role on strengthening the joint (Moore, 1992:475).

2.6.7. Movements at the hip joint

Movements at the hip include flexion, extension, adduction, abduction, internal and external rotation and circumduction (Moore, 1992:477).

2.6.7.1. Flexion

Reid, (1992:604) described the range of motion of hip flexion to be approximately 140°. The motion is limited by the soft tissue apposition of the thigh on the abdomen. Hip flexion is diminished to approximately 90° when the knee is placed in the extended position. This is due to an increase in hamstring tension. In the trained athlete this figure can increase. The maximum force of contraction is at approximately 30°-35° of hip flexion (Reid, 1992:605).

2.6.7.2. Extension

Only about 10° to 20° of true extension occurs at the hip. The illiofemoral and ischiofemoral ligaments and the Iliopsoas muscles limit this movement to such a narrow range. The muscles generate their greatest force at approximately 40° to 45° short of the midline (Reid, 1992:607).

2.6.7.3. Abduction

The action of hip abduction is limited by muscle tightness in the adductor muscles and via the pubofemoral ligament and medial aspect of the illiofemoral ligament. The range of motion is approximately 50°, until the pelvis begins to tilt to prevent impingement of the greater trochanter. However, anatomically the joint can allow up to 90° of motion, which can be achieved by abducting the leg when it is in the laterally rotated position (Reid, 1992:608).

During abduction, maximum power is achieved in the neutral position and decreases as it moves through this motion (Reid, 1992:608).

2.6.7.4. Adduction

The opposite leg limits hip adduction. With the contra-lateral leg flexed, 40° of adduction is possible. This motion is limited by the lateral band of the illiofemoral ligament and the ligament of the head of the femur (Reid, 1992:609).

2.6.8. Muscles responsible for movement at the hip

The table below gives a summary of the muscles responsible for movement at the hip and their respective segmental nerve supply.

Table 2.6. Muscles of the hip joint

MOVEMENT AT THE HIP	MUSCLES RESPONSIBLE	SEGMENTAL NERVE SUPPLY
Flexion	**Iliopsoas** **Tensor fascia lata** **Rectus femoris** Pectineus Sartorius Adductor longus	**L1,L2,L3** **L4,L5** **L2,L3,L4** L2,L3 L2,L3 L2,L3,L4
Extension	**Gluteus maximus** **Semi-tendinosus** **Semi-membranosus** **Biceps femoris** Adductor magnus	**L5,S1,S2** **L5,S1,S2** **L5,S1,S2** **L5,S1,S2** L2,L3,L4
Abduction	**Gluteus medius** **Gluteus minimus** Tensor fascia lata Sartorius Piriformis Obturator externus	**L5,S1** **L5,S1** L4,L5 L2,L3 S1,S2 L3,L4
Adduction	**Adductor magnus** **Adductor longus** **Adductor brevis** Pectineus Gracilus	**L2,L3,L4** **L2,L3,L4** **L2,L3,L4** L2,L3 L2,L3

All the muscles shown in bold are the main contributors to the relevant actions. The muscles in normal font assist the actions.
(Moore, 1992:476; Moore and Dalley, 1999:540-560)

2.7. ISOKINETIC DYNOMOMETRY

2.7.1. Introduction to isokinetics

Isokinetic assessment has been primarily recommended for strength testing as a maximal force is applied during all phases of movement of the joint at a constant velocity (De Ste Croix *et al*. 2003).

Isokinetic exercise has been defined as a dynamic muscular contraction when the velocity of the movement is controlled and held constant by a specific isokinetic device, usually an electromechanical appliance (Chan and Maffulli, 1996:7). The patient can not exceed the speed that has been set by the machine, and the machine matches the amount of force exerted by the patient (Cybex. 1996:p1-9). Isokinetics is not a type of muscle contraction, rather a type of exercise. The types of muscle contractions produced during isokinetic muscle testing are either concentric or eccentric muscle contractions (Chan and Maffulli, 1996:7).

Concentric muscle contraction was defined by Chan and Maffulli (1996:5), as a shortening of the distance between the origin and insertion of the muscle as it contracts and develops tension. Conversely, the same authors defined eccentric muscle contraction as the lengthening between the origin and insertion of the muscle as it contracts. Eccentric actions have been found to generate greater muscular tension and require less muscle work than concentric actions (Lieber, 1992).

Although these contractions do not closely resemble the nature of most joints' movements during normal human movements, Isokinetic assessment is the main method that is available to investigate whether the static or dynamic properties of the muscle are intact or not (Arokoski, 2001).

Isokinetic exercise is able to quantify muscle function by evaluating peak torque, average torque, work and power of the muscle that is contracting (De Ste Croix, *et al.* 2003).

2.7.2. Reliability and validity of isokinetic testing

2.7.2.1. Reliability

The reliability of isokinetic dynamometers have been found to be extremely high, with the accuracy of peak torque, work and power showing correlation coefficients between 0.93 and 0.99 (Magnusson, Gleim and Nicholas, 1990). Callaghan, *et al.* (2000) conducted a study into the test–retest reliability of isokinetic testing. The study used a sample of patients suffering from patella femoral pain syndrome compared to a control group of healthy individuals. They were able to conclude from their study that isokinetic values for peak torque, average power and total work using a multi-joint attachment were highly reliable and should be used by clinicians with confidence.

According to Chan and Maffulli (1996) the hip joint has a lower reliability due to its large range of motion. Chan and Maffulli (1996) also concluded that concentric results showed a greater reliability than eccentric results.

2.7.2.2. Validity

In assessing the validity of isokinetic testing it is necessary to look at validity in different settings.

Isokinetic testing has been found to have content validity in specific aspects of muscle performance, meaning that the clinical setting of isokinetic muscle testing can in some instances be comparable to real life situations. An example of this is the maximum power derived from isokinetic testing during the action of plantar flexion is equal to the

functional velocity of toe off during walking (www.isokinetics.net, 2003).

Convergent validity has been established by recognising the relationship that exists between certain factors and isokinetic testing. These relationships include:

2.7.2.2.1. Gender differences

Men have been found to be significantly and consistently stronger than females (De Ste Croix, *et al.* 2003).

2.7.2.2.2. Effect of age

Increases in strength occur up until approximately the third decade. After the third decade there is a moderate decrease in strength from then on, until the seventh decade when the decrease in strength becomes more marked (De Ste Croix, *et al.* 2003).

2.7.2.2.3. Body weight

The relationship between body weight and isokinetic values has been shown to rise proportionately. The higher the body weight the higher the isokinetic value. This is thought to be caused by an increase in muscle mass causing an increase in body weight. However, this relationship is not linear (De Ste Croix, *et al.* 2003)

2.7.2.2.4. Motivational Factors

De Ste Croix *et al*. (2003) stated that a patients maximal effort whilst performing isokinetic testing was influenced by motivational factors like verbal encouragement and visual feedback. Patients were found to reach more of their maximum potential in the presence of motivational factors.

These relationships should be considered when assessing isokinetic test results as the relationships have an impact on the validity of the results.

In assessing the predictive validity of isokinetic testing in being able to predict injury, Eriksson (1991) found no relationship between pre-season isokinetic testing and the rate of injury during the season. Isokinetic testing has been proposed as a tool to predict the progress of rehabilitation, by being able to provide a general forecast of the speed of recovery, however the investigations into the predictive validity of isokinetic testing is very sparse (www.isokinetics.net, 2003).

2.7.3. Gravity correction

When performing isokinetic testing on a limb through a gravity dependant position it is necessary to incorporate gravity correction procedures. This will ensure that the movements against gravity are not under estimated and the movements with gravity are not over estimated (De Ste Croix, *et al.* 2003).

By incorporating these measures it will add validity to the results.

2.7.4. Advantages of isokinetic testing

Isokinetic exercise is an objective, reproducible and quantifiable assessment of a muscle's performance (Chan and Maffulli, 1996:10).

Isokinetic exercise is beneficial as it allows maximal dynamic loading of the muscle throughout the muscles range of motion. The loading of the muscle is done with a constant accommodating resistance that is set at a specific speed; this increases the safety of the exercise (Chan and Maffulli, 1996:7).

Due to the safety aspect of isokinetic exercise it plays an important role in early muscle rehabilitation. It also has the added advantage of being able to isolate weak muscle groups by strapping and limiting range of motion to a specific angle of movement (Cybex. 1996:1-10).

Isokinetic testing makes accommodation for the patient's pain and fatigue (Cybex. 1996:1-10).

2.7.5. Limitation of isokinetic exercise

An Isokinetic Dynamometer, which is the equipment needed to perform isokinetic exercise and evaluation on, is very expensive causing a restriction on the use of isokinetic exercise due to its affordability (Chan and Maffulli, 1996:8).

Isokinetic exercise needs to be performed under the supervision of a specially trained person and the results also need to be assessed by a trained person, this places further limitations on the use of isokinetics as a therapeutic tool (Chan and Maffulli, 1996:8).

A further limitation is caused by the non-specificity of functional training for the lower extremity in the closed kinematic chain fashion (Perrin, 1993:7).

2.7.6. Isokinetic dynamometry as a tool

Isokinetic testing is an important therapeutic tool in evaluation, rehabilitation, research, diagnosis of injury and a training aid for muscles (Chan and Maffulli, 1996:10).

The main function of isokinetics lies in the ability to evaluate muscle strength. It provides a wide range of information on dynamic performance variables of a muscle group and is able to quantify objective values for work, force and power (Chan and Maffulli, 1996:10). Isokinetic testing has been successfully employed in the measurement of strength in patients with sports injuries (Yeung *et al.* 1994).

2.7.7. Interpretation of isokinetic data

Isokinetic dynamometers measure angular velocity, the position of the moving body part and either force or torque (Chan and Maffulli, 1996:11).

The peak torque of concentric muscle contractions gives a good indication into the power output and work of the muscle being tested. The unit of measurement for peak torque is Newton's (force) per second [N/s] (Jackson, 2003).

2.7.8. Isokinetic testing at the hip

Isokinetic testing of the hip joint can be performed in both the saggital[17] and coronal[18] planes of movement (www.isokinetics.net, 2003).

Flexion and extension of the hip are performed in the saggital plane. The exact range of motion at the hip in the saggital plane is debatable. In a comprehensive study Cahalan, (1989) suggested that 45 degrees hip flexion is the point of maximum efficiency for evaluating flexion and extension. Consequently, strength measurements should be made from 0 degrees flexion to 75 degrees flexion, and back for extension.

Hip movements in the frontal plane constitute the actions of hip abduction and adduction. Adduction has an estimated range of motion of up to 25 degrees and is prevented by the mechanical block of the other leg. Abduction has an estimated range of motion of 45 degrees (Miller, 1985).

Isokinetic testing of abduction and adduction can be performed in either the upright or side lying positions. Caution should be taken to ensure the knee is kept straight at all times. The axis of rotation at the hip joint is taken as the greater trochanter. The greater trochanter of the hip is aligned with the axis of rotation of the dynamometer (www.isokinetics.net, 2003).

This action of testing causes concentric-concentric muscle contractions (Chan and Maffulli, 1996:7).

[17] The saggital plane is an imaginary plane dividing the body into left and right portions (Moore and Dalley, 1999:3)

2.7.9. 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, would provide a valuable tool for the evaluation of muscular capability and injury assessment, especially in the Sports Medicine setting (Pincivero, Lephart and Karunakara. 1997 and Clifton, 2003).

[18] The coronal plane is an imaginary plane dividing the body into front (anterior) and back (posterior) portions (Moore and Dalley, 1999:3)

2.8. SUMMARY

Arthrogenic muscle inhibition is a limiting factor in the rehabilitation of joint injury. It results in strength deficits often long after healing has occurred. Arthrogenic muscle inhibition prevents the injured subject from performing active exercise in order to help increase healing (Hopkins and Ingersoll, 2000). Investigations into the effective treatment of arthrogenic muscle inhibition are needed. The goal of these treatments should be to reduce or eliminate the presence of arthrogenic muscle inhibition (Ingersoll, Palmieri and Hopkins, 2003).

The stimulation of nociceptors as in the case of a symptomatic sacroiliac syndrome can lead to the development of arthrogenic muscle inhibition of the muscles within the joint's motorneuron pool (Hopkins and Ingersoll, 2000). Therefore, arthrogenic muscle inhibition originating at the sacroiliac joint will cause inhibition of the muscles responsible for actions at the hip, because these muscles are within the motorneuron pool of the sacroiliac joint (Moore and Dalley 1999:540).

One of the final outcomes of arthrogenic muscle inhibition is a decrease in voluntary contraction of the affected muscle. Assessing the voluntary force output (peak torque) of the muscle is an effective and simple measure to indicate the level of voluntary contraction (Hopkins and Ingersoll, 2000).

Isokinetic dynamometry has been shown to be a valuable and reliable tool for the assessment and evaluation of muscle performance, especially in measuring peak torque (Pincivero, Lephart and Karunakara, 1997).

Suter, *et al.* (1999) and Suter, *et al.* (2000) found evidence that manipulation stimulated joint receptors of the adjusted joint, altering the afferent innervation and consequently causing a reduction in arthrogenic muscle inhibition. From these studies and the supporting literature it seems plausible that manipulation could be beneficial in treatment of arthrogenic muscle inhibition.

This research therefore aims to investigate the effects of sacroiliac manipulation in reducing arthrogenic muscle inhibition at the hip. Assessment will be made by evaluating the hip muscle strength on an isokinetic dynamometer before and after the manipulation.

CHAPTER THREE

3. MATERIALS AND METHODS

3.1. INTRODUCTION

This chapter outlines the general procedures utilized in carrying out this study. This includes, study design, the subjects (patients) used and a detailed account of the interventions that they received. Measurements and observations obtained as well as statistical procedures for the assessment of data are also discussed.

3.2. THE STUDY DESIGN

This study was a quantitative, prospective randomised pre- post- investigation, as the subject's hip strength was compared to their themselves before and after manipulation.

3.2.1. THE SAMPLE SELECTION

Non-probability based, purposive sampling was used on patients that responded to advertisements (Appendix G) placed in and around the Durban Institute of Technology, Sports clubs, gyms, local health shops and clinics.

Respondents to the advertisements were initially assessed for suitability by telephonic interview. The respondents were asked about the character, location and duration of their low back pain, their age, sex and availability to be included in the study. This telephonic interview was a cursory interview to establish if they met the basic inclusion and exclusion criteria to be included in the sample. If the respondent was found to be suitable an initial appointment was scheduled for their earliest convenience.

3.2.2. THE SAMPLE SIZE

The sample was made up of the first thirty male respondents who were suffering from chronic Sacroiliac syndrome.

3.2.3 THE SAMPLE CHARACTERISTICS

Before the patients were included into the study they were given a letter of information (Appendix D), which detailed the nature and aim of the study and explained the implications of their involvement in this study. Patients were told they may withdraw from the study at any time without giving a reason for their withdrawal and without incurring repercussions for future treatment.

The patients were given an opportunity to ask any questions regarding the study, which were then answered by the researcher. When all questions were answered, the subjects' were asked to complete an informed consent form (Appendix E).

An initial consultation was conducted at the Durban Institute of Technology Chiropractic Day Clinic in order to ensure a specific diagnosis of chronic sacroiliac syndrome. This was done by means of a case history (Appendix A), physical examination (Appendix B) and lumbar regional examination (appendix C) in order to assess the participant for the following inclusion and exclusion criteria:

3.2.4. INCLUSION AND EXCLUSION CRITERIA

3.2.4.1. Inclusion Criteria

- Only subjects between the ages of 18-45 years were accepted into this study. Subjects older than 45 years were not included because Brandt (2002) found that little radiographic evidence of osteoarthritis existed in people below the age of 45 years.

- Patients were only accepted if they:
  - Had given informed consent and completed a Numerical Pain Rating Scale (Jenson, *et al.* 1986)(Appendix F).

❖ Patients had to have a chief complaint of low back pain that was attributable to a chronic Sacroiliac joint syndrome. The condition was considered chronic if it was present for thirteen or more weeks (Giles and Muller, 2003).

A diagnosis of sacroiliac syndrome was reached if:

- Pain was felt over the sacroiliac joint, with possible referred pain to the groin, trochanter and buttock (Riggien, 2003, McCullach and Transfeldt, 1997: 180-181 and Cox, 1998:735).

- The sacroiliac joint was locally tender to palpation (Riggien, 2003, McCullach and Transfeldt, 1997: 180-181 and Cox, 1998:735).

- There was clinical evidence of abnormal movement or asymmetry of the sacroiliac joint (Riggien, 2003, McCullach and Transfeldt, 1997: 180-181).

- There was no other apparent cause of the patient's sacroiliac joint pain localization i.e. infection (Riggien, 2003, McCullach and Transfeldt, 1997: 180-181 and Cox, 1998:735).

- The pain was aggravated by provocation tests, like Gaenslen's, Yeomans, Posterior shear and Patrick Faber tests (Riggien, 2003, McCullach and Transfeldt, 1997: 180-181 and Cox, 1998:735).

1. Posterior shear test (Laslett and Williams, 1994)

This test is also known as the *thigh thrust test.*

Mechanism

A posterior shearing stress is applied to the sacroiliac joint through the femur.

Procedure

This is achieved by the patient being positioned supine, with the affected sides' knee and hip flexed and slightly adducted. The doctor places his one hand under the affected sacroiliac joint and applies a downward, posterior shearing force through the knee and femur with his other hand. The hand placed under the sacroiliac joint feels for joint motion when this force is applied.

Result

A positive test result is pain over the sacroiliac joint being tested.

2. Gaenslen's test (Laslett and Williams, 1994)

This test is also known as the *pelvic torsion test.*

Mechanism
The joint is stressed at its end of range of motion.

Procedure
The patient is supine. The hip and knee on the suspected side of dysfunction are flexed, whilst the hip on the opposite side is extended simultaneously. The examiner applies overpressure on the thigh, causing the joint to be stressed at its end of range of motion. This position causes the illium on the affected side to rotate posteriorly on the sacrum.

Result
A positive result is pain over the joint being tested.

3. Patrick FABER test (Kirkaldy-Willis and Burton, 1992:125)

Mechanism
The sacroiliac joint is stressed at the joint's end of range of motion.

Procedure
The patient is supine, with the hip on the side of the sacroiliac joint to be tested placed in to the flexed, abducted and externally rotated position. The examiner then places his one hand over the opposite iliac crest and with his other hand pushes down on the medial aspect of the knee on the side being tested.

Result

A positive test result is established if this position causes pain over the sacroiliac joint being tested.

4. Yeoman's test (Kirkaldy-Willis and Burton, 1992:125 and Magee 2002:526).

This provocation test is sometimes called *Erichsons test.*

Mechanism

This test stresses the posterior sacroiliac joint.

Procedure

The patient is in the prone position. The examiner places one hand under the thigh above the knee, on the same side as the suspected sacroiliac syndrome. The other hand is placed on the crest of the illium. The examiner causes extension of the hip, by pulling up with the hand on the thigh and applying downward pressure with the hand on the iliac crest. This action causes stress in the joint.

Result

A positive result is obtained if this procedure reproduces pain over the sacroiliac joint being tested.

3.2.4.2. Exclusion Criteria

Subjects were excluded from the study if they had:

- Previous low back surgery, as this would weaken the supporting muscles and predispose the patients to recurrent low back pain. This would

compromise the validity of the study (Kirkaly-Willis and Burton, 1992: 386-391).

- Were on any medication or receiving any other form of treatment for their low back pain (Haldeman, 1992:641).

- A condition which would be contra-indicated[1] for spinal manipulation (Gatterman, 1990:84 and Bergmann, Petersen and Lawrence, 1993:132)
  - Disc herniations with increasing signs and symptoms of neurological deficit
  - Vascular anomalies e.g. Abdominal Aortic Aneurysm
  - Lumbar spine tumors e.g. Bone tumours or metastasis
  - Lumbar spine infections e.g. osteomyelitis
  - Lumbar spine traumatic injuries
  - Psychological overlay e.g. malingering, hysteria

- Patients were excluded if they showed any of the following contra-indications to isokinetic muscle testing : (Jackson, 2003 and www.isokinetics.net, 2003)

[1] Any symptom or circumstance denoting the inappropriateness of a form of treatment that would otherwise be advisable (Redwood, 1997:335).

Absolute

- Non united fractures to limb
- Epilepsy
- Cardiac insufficiency
- Severe peripheral vascular disease
- Aneurysm
- Anticoagulants
- Recent (less than 3 months) chemotherapy
- Long term steroid use (more than three months)
- Acute (less than 7 days) muscle/ligament tear (more than grade 1)
- Pregnancy
- Severe osteoporosis
- Malignancy in the area to be tested

Relative

- Pain
- Severe limited range of motion
- Effusions
- Soft tissue or bone healing

❖ Female patients were excluded from this study due to the anatomical differences between the male and female pelves, which affect the hip joint

(Moore, 1992:247). Females were also excluded because of the differences in strength between the genders, with females averaging two-thirds the strength of males (Brukner and Kahn, 2002: 677). Excluding females helped to ensure a homogenous sample group and therefore increased the validity of the study.

- Patients who exhibited any of the above exclusion criteria were excluded. All drop-outs were replaced.

3.3. THE METHOD

Once a diagnosis of chronic sacroiliac syndrome had been reached the subjects were included in the study and a second appointment was scheduled. No treatment for the subjects' low back pain was given at the initial consultation.

The second appointment took place with a biokineticist at the Medigate Medical centre in Umhlanga Rocks. This appointment was scheduled at the convenience of the patient and the biokineticist. At this appointment the subjects underwent isokinetic testing of their hip on the same side as the sacroiliac syndrome. The patient then received a manipulation of the affected sacroiliac joint. After the manipulation the patient was asked to re-perform the isokinetic testing of the same hip.

3.4. INTERVENTION

The study contained no placebo or control group. All subjects were placed into one group that received the same intervention (sacroiliac manipulation) and identical methods of objective (isokinetic muscle testing on a cybex dynamometer) and subjective measurements (numerical pain rating scale).

3.4.1. Sacroiliac manipulation

3.4.1.1. Sacroiliac restrictions

Restrictions in the sacroiliac joint were assessed using motion palpation (Harrison, Harrison and Troyanovich, 1997) and Gillet's test (flexed-knee-raising test) (Sturesson, Uden and Vleeming, 2000). Both these authors questioned the reliability of motion palpation and Gillet's test respectively in the diagnosis of sacroiliac syndrome. For the purpose of this study these tests were not used in confirming diagnosis but rather as aids in detecting restrictions.

If patients presented with a bilateral sacroiliac syndrome they were asked to make a subjective decision as to which side was more symptomatic, only the side chosen was treated (Suter, *et al.* 2000). The effects of arthrogenic muscle inhibition can be seen on the contra-lateral side (Suter, *et al.* 1998), therefore a

bilateral sacroiliac syndrome could cause additional arthrogenic muscle inhibition on the side being tested (due to the contribution from the contra-lateral leg). This study, however only investigated the ipsilateral effects of arthrogenic muscle inhibition and sacroiliac manipulation.

Restrictions in the sacroiliac joint can be found in either the upper or lower aspects of the joint. The joint dysfunction could be either in the movement of flexion or extension. The sacroiliac joint was only manipulated according to the restrictions that were detected (Schafer and Faye, 1990).

3.4.1.2. Manipulative procedure

Patient position

The patient was set-up in the lateral recumbent position with moderate torso rotation. The upper leg was flexed at the hip just short of 90°. The patient's arms were folded on the patient's chest (Szaraz, 1990).

If the patient had a flexion fixation the lesion side was up (i.e. the lesion side was not making contact with the table in the lateral recumbent position). If the patient had an extension fixation then the patient was positioned with the lesion side down (i.e. the side of the lesion was in direct contact with the table when the patient was in the lateral recumbent position) (Szaraz, 1990, Schafer and Faye, 1990).

Doctor position

The doctor was standing in a fencer's stance with contact of his caudad hand on the sacroiliac joint over the restriction. The doctor's cephalid hand stabilized the patient's upper body at the shoulder. Stress was initiated into the sacroiliac joint at the point of the restriction. A body drop thrust was applied by the doctor when the elastic barrier was met (Szaraz, 1990).

Protocol for manipulation

The manipulation consisted of a high velocity, low amplitude thrust. The line of drive was in an inferior direction (Szaraz, 1990).

The Doctor was given only one thrust per adjustment. An audible cavitation[2] was not necessary to indicate a successful adjustment (Suter 1994).

3.5. THE DATA COLLECTION PROCEDURE AND MEASUREMENTS

Subjective measurements were obtained using the numerical pain rating scale 101 (Appendix F) (Jenson *et al.* 1986). These measurements were obtained at the initial consultation and at the end of the second appointment.

Objective measurements were taken with a Cybex Orthotron II Isokinetic Rehabilitation System. The Cybex dynometer was utilized to measure the torque of the muscle. Readings were obtained during hip flexion, extension, abduction and adduction both pre- and post manipulation.

3.5.1. THE DATA

The data used in this study was both primary and secondary data.

3.5.1.1. The Primary Data

a) Objective Data

The objective data was obtained using a Cybex Orthotron II Isokinetic Rehabilitation System at the Medigate Medical Centre in Umhlanga Rocks. The Cybex Dynamometer was used to measure the peak torque

[2] The effect of a manipulation associated with an audible release (Kirkaldy-Willis and Burton, 1992:288).

during concentric muscle contractions of the hip, whilst performing the actions of hip flexion, extension, adduction and abduction.

b) Subjective Data

Subjective data was obtained directly from the subjects. The subjects were asked to complete a numerical pain rating scale at the initial consultation and after completion of the second appointment. This was used to assess for any improvement in the subjects subjective perception of pain.

3.5.1.2. The Secondary Data

The secondary data utilised in this study was in the form of recent journal articles, books and related internet sites. This Data was found in the libraries of the Durban Institute of Technology and the University of Natal Medical school.

3.5.2. MEASUREMENTS

3.5.2.1. Objective measurements

The peak torque of muscle contraction during **isokinetic testing** was used as the study's objective measurements.

The subjects underwent concentric-concentric isokinetic muscle testing of the hip in the actions of flexion, extension, adduction and abduction. The isokinetic testing was set at a velocity of 60° per second.

Computerized gravity correction was used in order to eliminate confounding errors due to the weight of the limb being tested (Chan and Maffulli, 1996: p16 and Pincivero, Lephart and Karunakara, 1997).

The Cybex machine was calibrated weekly during the research to ensure the readings were accurate.

All subjects received similar, equally enthusiastic verbal encouragement during the testing to ensure their maximal effort.

Patient Procedure

- 5-minute warm up cycle on a stationary bike
- Stretching of the muscles being tested. Stretching involved 3 repetitions held for 20 seconds each of the:

  → Iliopsoas

  → Quadriceps

  → Hamstrings

  → Adductors

  → Abductors

Stretching was only performed on the affected side.

- The subject then commenced isokinetic testing on the Cybex Dynamometer.

Patient Positioning

During isokinetic testing the actions of hip flexion and extension were tested in the same patient position **(patient position 1)**. Likewise, the actions of hip adduction and abduction were tested in the same patient position **(Patient position 2)**.

For **patient position 1** (Flexion and extension) the subjects were positioned:

- In the supine position to allow maximal hip flexion, with the affected side next to the power arm of the Cybex machine. Testing began with the patient in the fully flexed position (thigh at more than 90° to the table).

- A strap was used to anchor the patient over the abdomen to isolate movement at the hip joint.

For testing in **patient position 2** (Adduction and abduction) the subjects were positioned:

- In the lateral-recumbent position facing toward the power arm of the cybex dynamometer, with the affected side on top. This position allows the leg to freely perform the actions of hip adduction and abduction.

- A strap was used to anchor the patient over the torso to help isolate the movement to the hip joint.

For testing in both patient positions,

- The axis of movement of the power arm of the cybex dynomometer was aligned with the axis of movement of the hip joint at the femoral head.

- The length of the power arm of the cybex dynomometer was adjusted to incorporate the entire length of the femur and was secured (by straps) to the patient just proximal to the knee joint at the distal femur.

- The subject was told not to hold onto the machine or to gain leverage of the upper body to assist in performing the actions being tested.

Testing Procedure

The following testing procedure was followed for both isokinetic muscle testing positions:

- 2-3 sub-maximal warm-up repetitions of the actions being tested, at 60° per second.
- 1 minute rest.
- 1-2 trial repetitions of maximal effort at 60° per second.

- 1-minute rest.
- 3 test repetitions of maximal effort at 60°per second.

A 1-2 minute rest was given to the patients after completion of testing in **patient position 1** before initiating testing for **patient position 2**.

Subjects then received an ipsilateral sacroiliac manipulation.

Immediately after the manipulation the subjects re-performed the same isokinetic testing procedure on the same hip.

3.5.2.2. Subjective measurements

Subjective measurements were made using the numerical pain rating scale 101 (Jenson *et al.* 1986). This involved the subjects giving a number between 1 and 10 that best described the pain when it was at its least and another number between 1 and 10 that best described the pain when it was at its worst. A mean was then calculated between these two values.

Subjects were asked to complete two numerical pain rating scales. One during the initial consultation and one after the manipulation, during the second consultation. The means from these two tests were compared to assess for a subjective change in the subjects' perception of pain after the manipulation.

3.6. ETHICS

All subjects participated in the study voluntarily and received no remuneration for their involvement. After being properly informed about the study and having had all their questions regarding the study answered, patients were asked to give their written informed consent (Appendix E).

All the patients' information was regarded as strictly confidential and treated accordingly.

3.7. STATISTICAL ANALYSIS

3.7.1. Inter-group comparison

Inter-group analysis was done on hip strength for the four different actions being tested (flexion, extension, adduction and abduction). Inter-group analysis will evaluate whether one of the actions being tested responded more effectively or differently to the intervention given (manipulation).

The objective tests that were applied were the ANOVA test followed by the independent paired T-tests.

3.7.2. Intra-group comparison

Intra-group objective analysis of the data was performed on each of the hip actions being tested (flexion, extension, adduction and abduction). Intra-group analysis was evaluated for each action for a statistically significant change in isokinetic strength results before and after the manipulation. Each action was assessed individually, independently of the other actions.

The tests that were applied were the:

1. Objective: Paired T-test.
2. Subjective: Paired T-test.

The level of significance was set at α = 0,05 and the p-values were used for decision making. All data was analyzed using the SPSS package (version 9). This statistical software program is manafactured by SPSS Inc, 444N. Michigan Avenue, Chicago, Illinois, USA.

Various descriptive and inferential statistical techniques were used. Descriptive statistics included various tables and graphs that used means, proportions and percentages of the data. Inferential statistics made use of hypothesis-testing techniques.

CHAPTER FOUR

4. RESULTS

4.1. Introduction

This chapter is concerned with the demographic data of all the participants included in this study. It also contains a detailed statistical analysis of the subjective and objective data collated throughout the duration of the study.

4.2. Index

A list of statistical abbreviations that will be used in this chapter:

(n = 30): The sample size of the study was thirty (30) male subjects.

This sample size allowed for the application of the central limit theorem and the use of stronger, more robust parametric testing techniques.

α: level of significance = 0.05

P-value: observed level of significance
The P-value is the smallest level of significance that would lead to the rejection of the null hypothesis.

H_0: Null hypothesis
States that there is no significant difference between the two variables that are being compared.

H_1: Alternate hypothesis
States that there is a significant difference between the two variables that are being compared.

If the P-value reported was less than 0.05 (level of significance), a significant result was declared and the null hypothesis (H_0) was rejected.

H_0 was rejected and H_1 was accepted if $P<\alpha= 0.05$
H_0 was accepted and the H_1 rejected if $P> \alpha= 0.05$

4.2.1. The Sub-problem

The objective was to evaluate if an immediate subjective or objective change in hip strength was observed after an ipsilateral sacroiliac manipulation in patients suffering from chronic sacroiliac syndrome.

The hypothesis for the study were as follows:

H_0: There was no difference in the comparison of observations from pre-manipulation to post-manipulation.

H_1: There was an increase between the observations when comparing the pre-manipulation values to post-manipulation values.

4.3. Demographic Data

There was only one group of 30 subjects. All participants were male.

4.3.1. Race Demographics

The sample consisted of 18 Whites (60%), 7 Blacks (23%), 2 Indians (7%) and 3 Coloreds (10%).

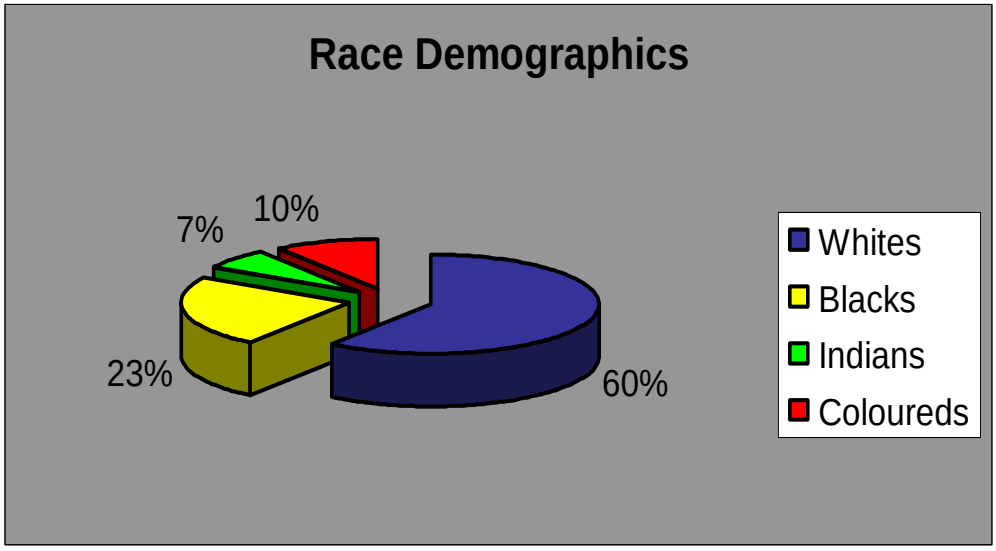


Figure 4.1. Race Demographics

4.3.2. Age Distribution.

The mean age was 28 years.
The age range was 19 – 44.

Figure 4.2. Age distribution of the sample

4.3.3. Height and Weight

The mean height of the sample was 1.77 meters.

The mean weight of the sample was 80.16 Kg's

The height range was 1,6 – 1,9 m.

The weight range was 55 – 118 Kg.

4.3.4. Occupation

The table below lists the occupations of the sample group.

Table 4.1. List of occupations

Occupation	Number of subjects
Accountant	1
Chef	3
Cleaner	1
Computer technician	1
Businessman	3
Entertainer	1
Journalist	1
Manager	1
Office assistant	1
Sales representative	1
Student	15
Umpire	1

4.4. Results of Statistical Analysis

The statistical results of the study were obtained by using the SPSS package. Statistical analysis involved both intra- and inter- group analysis of the data.

4.4.1. Inter-group Analysis

Inter-group analysis was performed to evaluate if one specific observation (actions and NPRS) responded more effectively than other actions. The objective ANOVA test was used to perform the inter-group analysis.

Table 4.2. ANOVA test results

ANOVA test	Mean Square	P- value
Analysis between observations	1.155	.726

The result was found to be statistically insignificant as the P-value (0.726) was greater than the level of significance (α = 0.05).

This result indicated that when comparing the results of the observations there was not one observation that was statistically more responsive than the others.

The follow-on paired T-tests were not conducted as the ANOVA test had an insignificant result, making these further tests redundant.

4.4.2. Intra-group Analysis.

Intra-group statistical analysis was performed on each individual observation to assess if a statistically significant change was observed when comparing pre-manipulation values to post-manipulation values.

4.4.2.1. Analysis of numerical pain rating scale 101 (NRS 101)

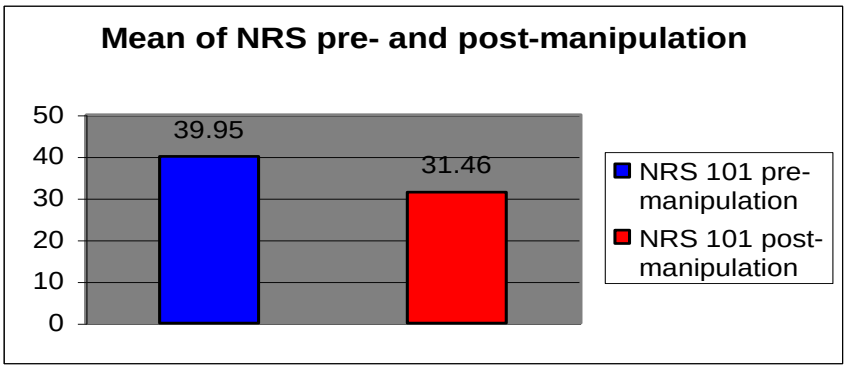


Figure 4.3. Comparison of NRS results

The mean difference between the pre-manipulation and post-manipulation results is a decrease of 8.49 points.

Table 4.3. Statistical comparison of NRS values

	Mean	Std Deviation	P-value
NPRS Pre vs Post	8.483	9.589	0.000

The Null-hypothesis was rejected for the comparison of the numerical pain rating scale pre-manipulation compared to the numerical pain rating scale post-manipulation. The P value (0.000) was less than 0.05, so the results were found to be statistically significant.

Analysis of hip flexion isokinetic strength results

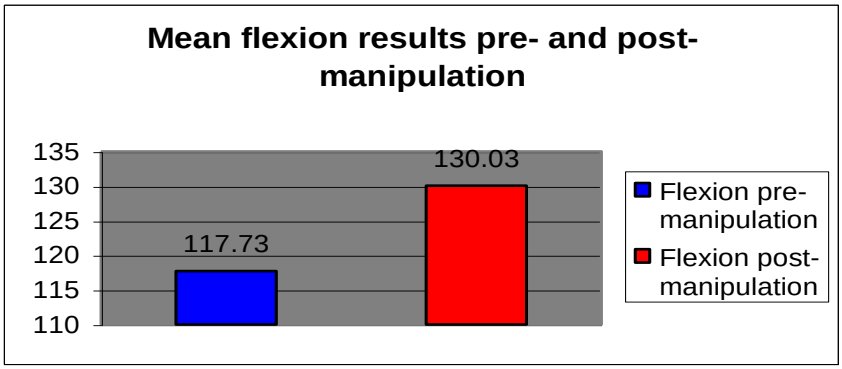


Figure 4.4. Comparison of flexion results

The action of hip flexion showed a mean increase of 12.30 isokinetic units when comparing the pre-manipulation value with the post-manipulation value.

Table 4.4. Statistical comparison of flexion results

	Mean	Std Deviation	P-value
Flexion Pre vs Post	-12.30	10.76	0.000

The null hypothesis was rejected for the comparison of hip flexion pre- and post- manipulation. The increase in hip flexion strength was found to be statistically significant as the P-value (0.000) was less than the level of significance ($\alpha = 0.05$).

4.4.2.3. Analysis of hip extension isokinetic strength results

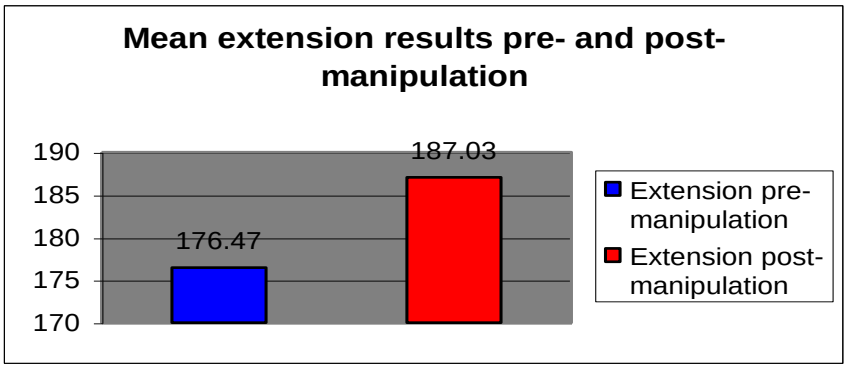


Figure 4.5. Comparison of extension results

The action of hip extension showed a mean increase of 10.56 isokinetic units when comparing the pre-manipulation value with the post-manipulation value.

Table 4.5.Statistical comparison of extension results

	Mean	Std Deviation	P-value
Extension Pre vs Post	-10.57	30.40	0.067

The null hypothesis was accepted for the analysis of hip extension strength before and after manipulation. An increase was observed, however this increase was not statistically significant. The P-value (P= 0.067) was greater than the level of significance (α = 0.05).

4.4.2.4. Analysis of hip abduction isokinetic strength results

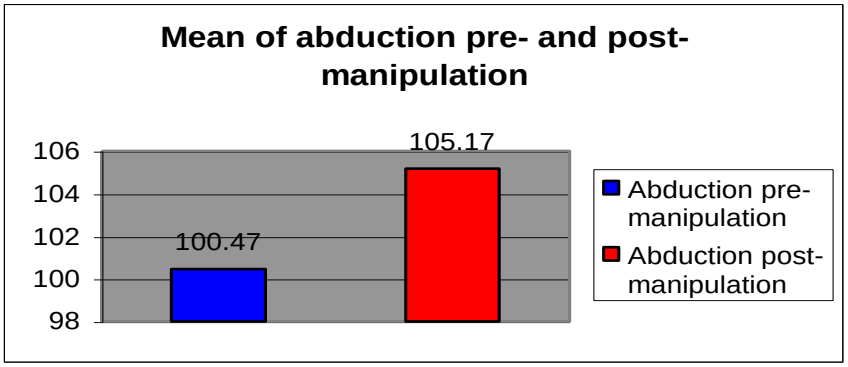


Figure 4.6. Comparison of abduction results

The action of hip abduction showed a mean increase of 4.70 isokinetic units when comparing the pre-manipulation value with the post-manipulation value.

Table 4.6. Statistical comparison of abduction results

	Mean	Std Deviation	P-value
Abduction Pre vs Post	-4.70	12.39	0.047

The null hypothesis was rejected because the P-value (P= 0.047) was found to be less than the level of significance (α = 0.05). The increase in isokinetic strength for the action of hip abduction was found to be statistically significant.

4.4.2.5. Analysis of hip adduction isokinetic strength results

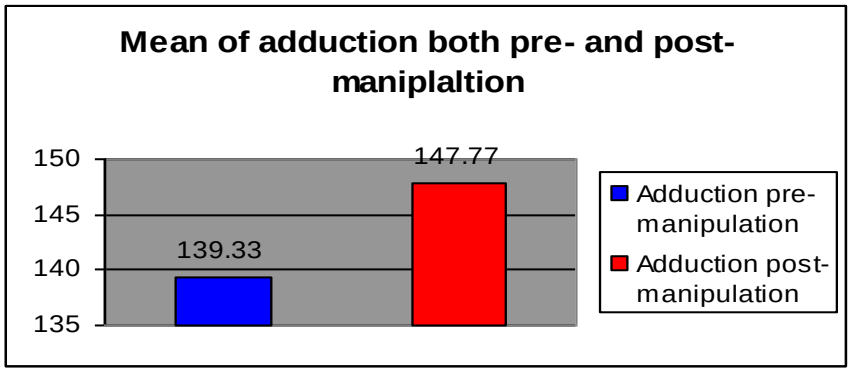


Figure 4.7. Comparison of adduction results

The action of hip adduction showed a mean increase of 8.44 isokinetic units when comparing the pre-manipulation value with the post-manipulation value.

Table 4.7. Statistical comparison of adduction results

	Mean	Std Deviation	P-value
Adduction Pre vs Post	-8.43	18.61	0.010

The Null hypothesis was rejected for the comparison of hip adduction. The P-value (0.10) was found to be less than the level of significance (α = 0.05). The increase in isokinetic strength for the action of hip adduction was found to be statistically significant.

4.5. Outcomes

Subjective Results

Numerical pain rating scale decrease in pain that was statistically significant

Objective Results

Flexion increase that was statistically significant
Extension increase, but was not statistically significant
Abduction increase that was statistically significant
Adduction increase that was statistically significant

A relationship between the subjective and objective data exists. The subjective perception of pain decreases whilst the objective gains in strength increase. Generally all results reached a statistically significant level.

These findings are in keeping with the removal of arthrogenic muscle inhibition.

CHAPTER FIVE

5. DISCUSSION OF RESULTS

5.1. Introduction

This chapter is concerned with the discussion and interpretation of the statistical results from the output in Chapter four.

Subjective data was obtained using the numerical pain rating scale 101. Objective data was derived from the results of the isokinetic strength testing.

Intra and Inter group statistical analysis was then performed on the data groups.

The initial part of this chapter will deal with the analysis of the data according to demographics. The latter part will deal with the interpretation of the inter- and intra-group statistical analysis.

5.2. Demographics

The different actions will be analyzed according to race, age, height and weight to observe for any relationships that may exist.

5.2.1. Race

a) Subjective data analysis

Analysis of the numerical pain rating scale between the different races showed a marked improvement amongst Whites and Indians, there was a smaller improvement in the black population. The colored community showed no subjective improvement of their pain. A possible reason for the lack of improvement by the colored population could possibly be attributed to a general lack of exposure of this community to chiropractic care, causing the colored population to be unfamiliar with manipulation. This rationale does however, have no validity and is an observation made by the researcher.

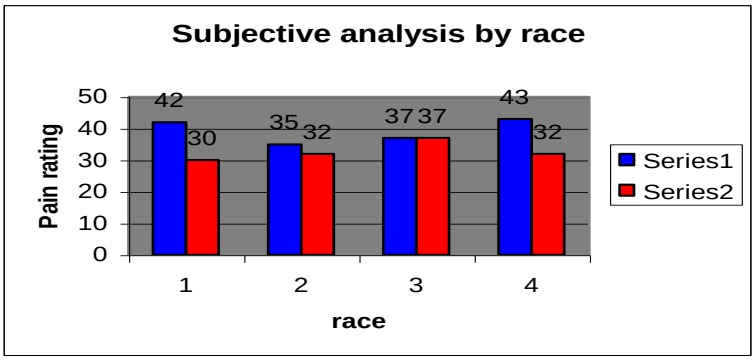


Figure 5.1. Subjective analysis by race

1= White, 2= Black, 3= Colored, 4= Indian.

Series 1 (Blue) = Pre- manipulation reading.

Series 2 (Red) = Post-manipulation reading.

b) Objective Data analysis

Analysis of the objective data according to race showed that the colored population responded the most to the intervention with all but one of the actions showing an increase, the action of adduction did not increase but remained the same.

These findings do not seem in keeping with the results of the subjective analysis, where the colored population showed the least improvement.

The low response of the colored population to the subjective analysis of pain could show that this group had a high perception of pain, when compared to the other populations. Arthrogenic muscle inhibition is caused by pain stimuli in the joint, so a higher level of perceived pain could cause a higher degree of arthrogenic muscle inhibition to be present in the joint. The manipulation would reduce the presence of arthrogenic muscle inhibition allowing for the observed increases of strength in this population.

The objective results of the colored population showed a good response to manipulation. The White population was the next responsive, followed by the Indian population. Least responsive was the Black population, which only showed an increase in the action of flexion. The numerical pain rating scale may be dependent on physiological mediators, which will take longer to resolve than the neurological response of arthrogenic muscle inhibition.

Table 5.1. Results based on race

Race	Flexion			Extension			Abduction			Adduction		
	Pre	Post		Pre	Post		Pre	Post		Pre	Post	
White	127	142	↑	184	199	↑	115	109	↓	144	157	↑
Black	101	109	↑	167	166	↓	83	76	↓	136	135	↓
Colored	118	134	↑	200	225	↑	120	121	↑	155	155	→
Indian	90	92	↑	111	99	↓	76	74	↓	84	100	↑

5.2.2. Age

a) Subjective analysis of data

Analysis of the numerical pain rating scale according to the different age groups showed that all the age groups had a decrease in their perception of pain across the age spectrum.

For the following two graphs:

Series 1 (blue) = pre-manipulation

Series 2 (red) = post-manipulation

1 = 18 to 21 yrs

2 = 22 to 25 yrs

3 = 26 to 29 yrs

4 = 30 to 33 yrs

5 = 34 to 37 yrs

6 = 38 to 41 yrs

7 = 42 to 45 yrs

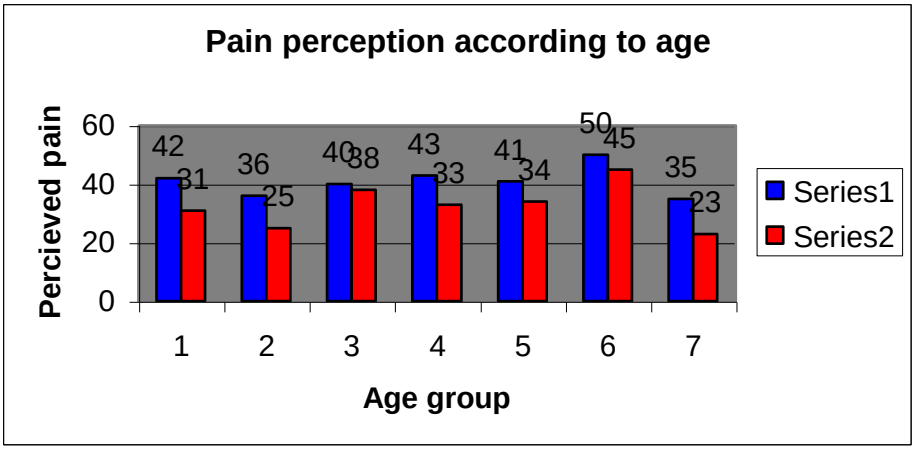


Figure 5.2. Pain perception according to age

b) Objective Data Analysis

All four of the actions being tested showed a uniform response when analyzed according to age. The majority of age groups showed an increase in the post-manipulation readings when compared to the pre-manipulation readings. The lower and higher age groups showed tapering isokinetic strength results.

Age has been found to exert an independent effect on strength development, most cross-sectional studies of isokinetic strength have demonstrated a significant increase in strength with age in males and females. This increase in strength usually occurs until the fifth decade then starts to decline every decade there after (De ste Croix *et al*., 2003). This explains the tapering of isokinetic values on either side of the age range observed in the results from this study.

The graph below illustrates the flexion isokinetic results according to age. The graph is indicative of the other actions that were tested (extension, adduction and abduction).

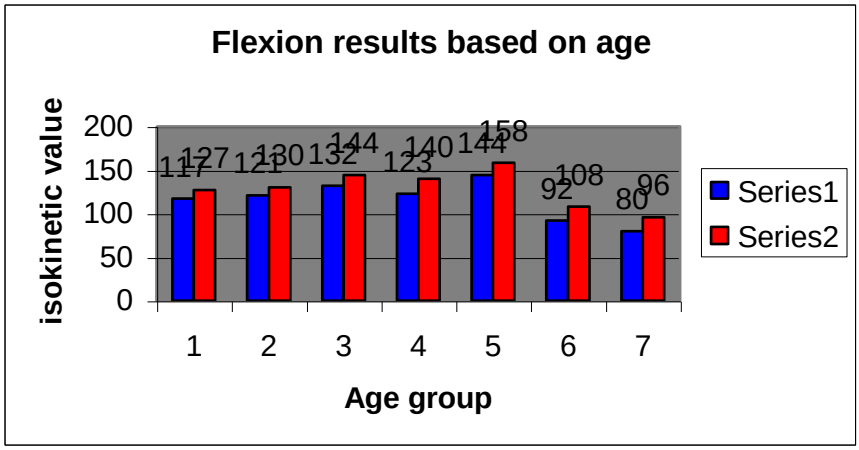


Figure 5.3. Flexion results based on age

5.2.3. Weight

a) Subjective Data Analysis.

Analysis of the subjective data according to weight did not show any correlations or relationships. There was a general decrease in pain perception across the sample in spite of differences in weight.

b) Objective Data Analysis.

The objective results are in keeping with Chan and Maffulli (1996) and www.isokinetics.net (2003) who reported how isokinetic testing values were directly proportional to the subjects height and body weight i.e. the higher the body weight and height the higher the isokinetic test value.

For All graphs:
Series 1 (Blue) = Pre-manipulation.
Series 2 (Red) = Post-manipulation

1 = 55 to 64 kg
2 = 65 to 74 kg
3 = 75 to 84 kg
4 = 85 to 94 kg
5 = 105 to 114 kg
6 = 115 to 124 kg.

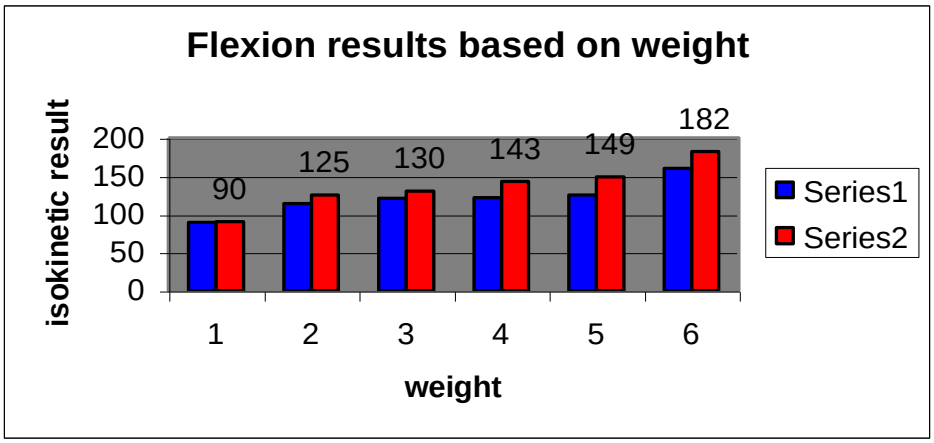


Figure 5.4. Flexion results based on weight

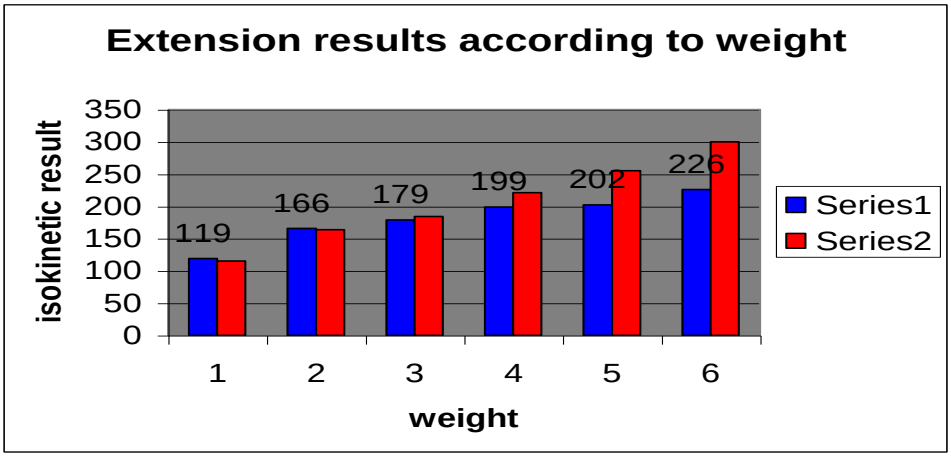


Figure 5.5. Extension results according to weight

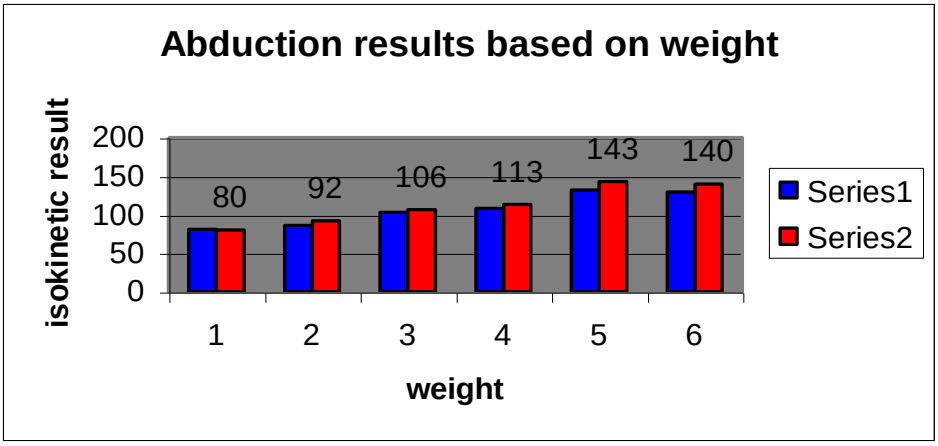


Figure 5.6. Abduction results based on weight

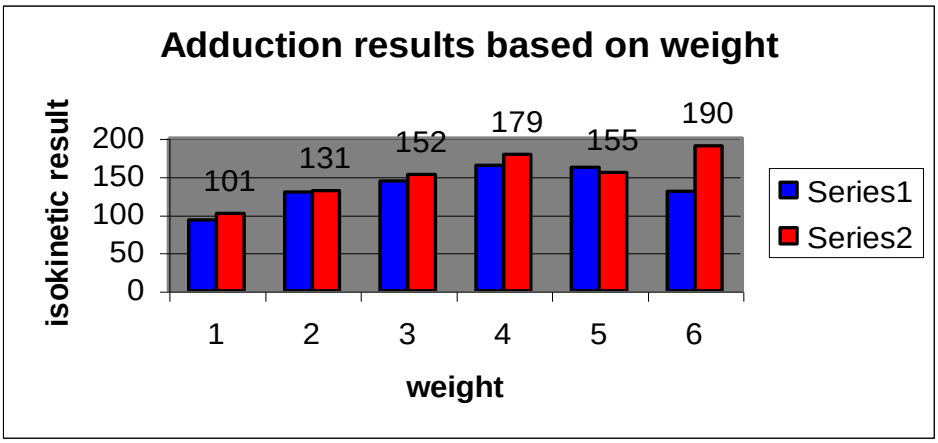


Figure 5.?. Adduction results based on weight

De Ste Croix *et al.* (2003) indicated that a strong relationship exists between stature, body mass and isokinetic leg strength. Both stature and mass independently influence isokinetic testing. A higher body mass could be due to a larger muscle mass, which would result in higher muscle torque and larger isokinetic strength readings.

The relationship between isokinetic hip strength and body mass can be observed in the preceding graphs by the higher isokinetic strength readings in the larger weight groups for each action.

The isokinetic values for hip flexion, extension, adduction and abduction increase as the weight of the group's increase. The majority (20/24: 83%) of the weight groups demonstrated an increase in the post-manipulation reading when compared to the pre-manipulation reading.

An interesting observation that can be made from the graphs is how the pre-manipulation values begin to stagnate in the larger weight groups. The actions of extension (figure 5.5.) and abduction (figure 5.6.) show marked stagnation with adduction (figure 5.?.) demonstrating a decline in pre-manipulation levels. This stagnation or decline could be due to the presence of arthrogenic muscle

inhibition, causing a decrease in strength, or not allowing the muscles to reach its' full potential. The reason for the evidence of the effect of arthrogenic muscle inhibition only being present in the larger weight groups could be attributed to the larger isokinetic strength values making the effect more evident.

If manipulation is believed to cause stimulation of joint mechanoreceptors and reduce arthrogenic muscle inhibition (Suter *et al.*, 2000), then the post-manipulation readings lend further support to arthrogenic muscle inhibition being the cause of the pre-manipulation stagnation. A post-manipulation increase in hip strength is observed in most (20/24) of the actions for all the weight groups. Of interest is the removal of the pre-manipulation stagnation. The post-manipulation readings show a return to the normal trend of the larger weight groups being associated with higher isokinetic strength values. These effects indicate the removal of arthrogenic muscle inhibition, probably as a result of the manipulation.

The restoration of the normal *body mass: isokinetic strength relationship* in the post-manipulation readings can be seen in flexion (figure 5.4.), extension (figure 5.5.) and the reversal of the downward trend in the action of adduction (figure 5.?.).

A return to the normal trends of isokinetic exercise as seen by these results lends validity to the objective results as they conform to the normal trends of isokinetic testing.

5.2.4. Height

Subjective and objective analysis of the data according to height produced no obvious relationship. All of the reading improved though.

5.3. Statistical Analysis

5.3.1. Inter-group statistical analysis

Inter-group statistical analysis was performed on the data using the one-way ANOVA test. The test was used to establish if one specific action responded more favorably than the other actions to a level that was statistically significant. The level of significance was set at $\alpha = 0.05$. The ANOVA test produced a result of P= 0.726. This level was higher than α, so the result was statistically insignificant. Meaning that there was not one action that out performed the other actions, all actions responded to the intervention to a similar degree. The results although not identical showed a similar trend.

The removal of sacroiliac arthrogenic muscle inhibition would cause an increase in all four of the hip actions because they all fall within the motorneuron pool of the sacroiliac joint. The effects of a manipulation in reducing arthrogenic muscle inhibition would cause an increase of strength in all the hip actions. The trend observed in the inter-group statistical analysis is in keeping with the removal of arthrogenic muscle inhibition as all the actions were expected to have increased strength after the removal or reduction of sacroiliac arthrogenic muscle inhibition.

5.3.2. Intra-group statistical analysis

Intra-group statistical analysis was used to analyze the results of each individual action to assess how each action responded to the intervention (sacroiliac manipulation).

5.3.2.1. Flexion

Paired T-tests performed on the results for flexion showed a P-value of 0.000. This figure was less than the level of significance (α = 0.05). Meaning that the increase from the pre-manipulation values after the manipulation was statistically significant.

The rationale for the increase in the post-manipulation isokinetic strength values can be explained by the removal of arthrogenic muscle inhibition.

The action of hip flexion is performed chiefly by the iliopsoas, Tensor Fascia lata and rectus femoris muscles (Moore and Dalley, 1999:540).

The Iliopsoas muscle is innervated by the ventral rami of lumbar nerves L1-L3 .The tensor fascia lata is innervated by the superior gluteal nerve (L4 and L5). Rectus femoris muscle is innervated by the femoral nerve (L2, but chiefly L3 and L4) (Moore, 1992:386). Suter, *et al.* (2000) described the innervation of the anterior sacroiliac joint as being derived from the anterior (ventral) primary divisions of the L2 to S2 spinal segments. As described in chapter two the action of hip flexion is within the motorneuron pool of the sacroiliac joint, allowing arthrogenic muscle inhibition originating in the sacroiliac joint to cause inhibition of hip flexion.

The increase in the pre-manipulation, mean flexion isokinetic strength value (117.73), compared to the post-manipulation mean flexion value (130.03) can be attributed to the removal of arthrogenic muscle inhibition. These findings support the rationale proposed by Suter *et al.* (1999) that manipulation causes stimulation of joint receptors within the adjusted joint, causing an altered afferent innervation, resulting in a decrease in arthrogenic muscle inhibition.

Of the actions being tested all actions showed a post-manipulation increase. The level of statistical significance varied: extension P-value = 0.067, abduction P-value = 0.047, adduction P-value = 0.010. Flexion showed the highest level of statistical significance with P-value = 0.000.

A probable reason for flexion out-performing the other actions could be explained by examining the nerve supply. As mentioned earlier the iliopsoas muscle is a chief mover in the action of hip flexion. The innervation of this muscle is the ventral rami of L1, L2 and L3 (Moore and Dalley, 1999:540). The anterior (ventral) primary divisions of L2-S2 innervate the anterior aspect of the sacroiliac joint (Suter, *et al.* 2000). Both the iliopsoas muscle and sacroiliac joint receive direct innervation from the anterior spinal segments. Whereas the other actions share the same segmental nerve supply through motor and sensory nerves.

This similar innervation and direct supply from the spinal segments could cause the action of hip flexion to be more susceptible to arthrogenic muscle inhibition arising from a sacroiliac syndrome. The similar innervation allows for a more direct pathway with less involvement of sensory and efferent nerves. Likewise, the direct segmental nerve supply could allow for the effects of the manipulation to be more effective than compared with the other actions. This would account for the higher increase in post-manipulation results for flexion that were not matched by the other actions that do not share this same spinal nerve segmental supply.

5.3.2.2. Extension

The mean, pre-manipulation, extension value (176.47) increased when compared to the mean, post-manipulation, extension value (187.03). When statistically analyzed this increase was found to be statistically insignificant. The P-value was 0.067, which was higher than the level of significance ($\alpha = 0.05$).

The muscles responsible for the action of hip extension are the gluteus maximus, semi-tendinosus, semi-membranosus and biceps femoris. All muscles are innervated by the L5, S1, S2 nerve segments. The segmental nerve supply falls within the motor-neuron pool of the sacroiliac joint. Theoretically the action of hip extension should be affected by arthrogenic muscle inhibition that arises from the sacroiliac joint. This could explain the increase observed between the mean pre-manipulation and post-manipulation isokinetic values. The reason for the increase not reaching a statistically significant level is more difficult to explain, as all the other actions experienced a statistically significant increase.

A possible explanation could be the opposite explanation to that given for the good response of flexion. The muscle responsible for hip extension are all innervated by motor nerves

Gluteus maximus via the inferior gluteal nerve
Semi-tendinosis and semi membranosis via the tibial division of sciatic nerve
Biceps femoris via branches of the sciatic nerve

Muscles for hip extension are within the same motorneuron pool as the sacroiliac joint but receive this innervation via motor nerves (Moore and Dalley, 1999). The direct spinal innervation observed in hip flexion does not exist for hip extension. Therefore the development and treatment of arthrogenic muscle inhibition does not have the direct innervation of hip flexion. This could make the action of extension less responsive to manipulation due to the more complex and involved neural pathway.

Further to this, the isokinetic testing protocol and procedure used during this study could have made the subjects prone to muscle fatigue as the four actions were tested one after the other and immediately before and after the manipulation. This did not allow much time for the subjects to recover this could have affected the extension results from gaining their full potential.

5.3.2.3. Abduction

The mean, pre-manipulation, abduction value (100.47) increased when compared to the mean, post-manipulation, abduction value (105.17). When statistically analyzed this increase was found to be statistically significant. The P-value was 0.047, which was lower than the level of significance ($\alpha = 0.05$).

The muscles responsible for the movement of hip abduction (gluteus medius and minimus) are innervated by the superior gluteal nerve L5, S1 (Moore and Dalley, 1999:551). This segmental nerve supply falls within the segmental nerve supply of the sacroiliac joint, which is L2 to S2 (Suter, *et al.*, 2000). Therefore the action of hip abduction is within the motorneuron pool of the sacroiliac joint.

Due to the statistically significant outcome it could be accepted that the manipulation caused excitation of the joint receptors, altering the afferent joint innervation causing a reduction in arthrogenic muscle inhibition. The reduction of arthrogenic muscle inhibition would clinically manifest itself as an increase in strength of the affected motorneuron pool. An increase in isokinetic strength was observed for the action of hip abduction. Making it plausible that this increase in hip abduction strength was as a result of the removal of arthrogenic muscle inhibition.

5.3.2.4.Adduction

The mean, pre-manipulation, adduction value (139.33) increased when compared to the mean, post-manipulation, adduction value (147.77). When statistically analyzed this increase was found to be statistically significant. The P-value was 0.010, which was lower than the level of significance (α = 0.05).

The adductor group of muscles (adductor longus, adductor brevis and adductor magnus) are responsible for the movement of hip adduction and are innervated by the obturator nerve L2, L3, L4 (Moore and Dalley, 1999:540). This segmental nerve supply falls within the segmental nerve supply of the sacroiliac joint, which is L2 to S2 (Suter, *et al.*, 2000). Therefore the action of hip adduction is within the motorneuron pool of the sacroiliac joint.

Due to the statistically significant outcome it could be accepted that the manipulation caused excitation of the joint receptors, altering the afferent joint innervation causing a reduction in arthrogenic muscle inhibition. The reduction of arthrogenic muscle inhibition would clinically manifest itself as an increase in strength of the affected motorneuron pool. An increase in isokinetic strength was observed for the action of adduction. Making it plausible that this increase in hip adduction strength was as a result of the removal of arthrogenic muscle inhibition.

5.3.2.4. Arthrogenic Muscle Inhibition

The findings of this study lend support to the assumption of this study that a chronic sacroiliac syndrome will cause the development of arthrogenic muscle inhibition of the hip musculature.

5.3.2.5. Effects of cavitation[1] on the results

Of the sample of 30 subjects, 23 had an audible cavitation when their sacroiliac joint was manipulated, whilst 7 of them did not. An audible cavitation was not necessary to indicate a successful manipulation (Suter, 1994). A graphic illustration of the results according to cavitation follows.

Series 1 (blue) = Pre- manipulation values
Series 2 (red) = Post-manipulation values
1 = **Cavitation**
2 = **No cavitation**

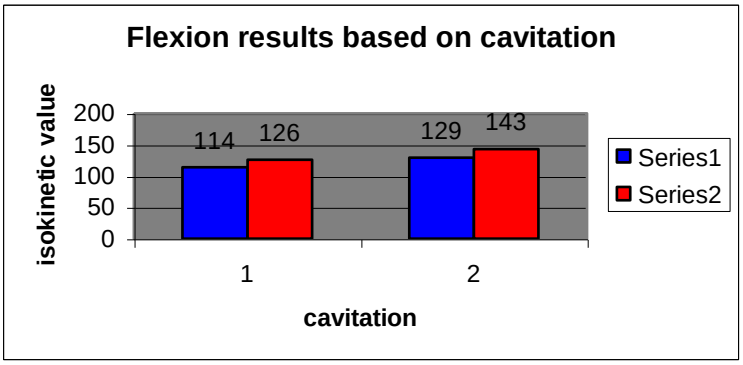
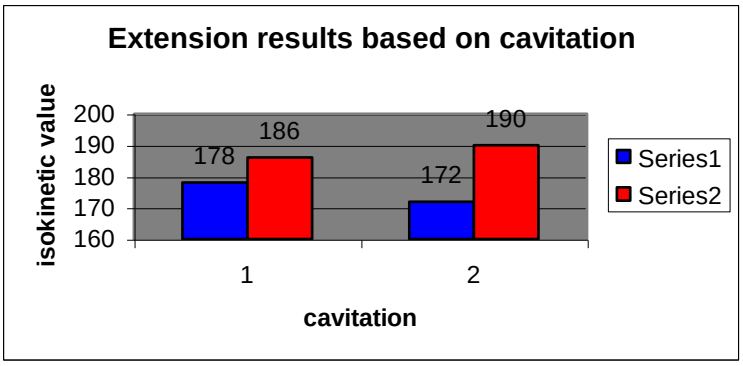


Figure 5.8. Flexion results based on cavitation

[1] The effect of a manipulation associated with an audible release (Kirkaldy-Willis and Burton, 1992:288)



5.9. Extension results based on cavitation

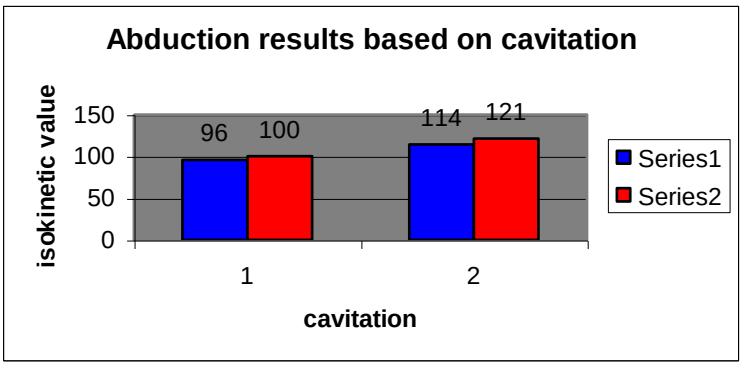


Figure 5.10. Abduction results based on cavitation

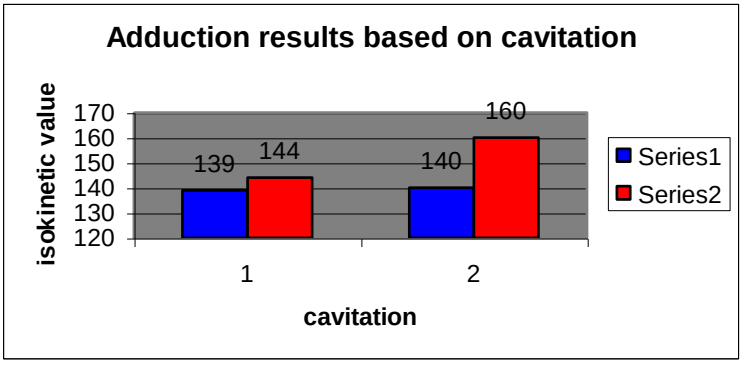


Figure 5.11. Adduction results based on cavitation

The effects of the manipulation were not dependent on an audible cavitation. The results from the non-cavitation group out performed the cavitation group. The table below tabulates the mean differences between the pre- and post-manipulation values for each action in the cavitation and non-cavitation groups.

Table 5.2. Results based on cavitation

Action	Cavitation	Non-cavitation	Difference
Flexion	12	14	2
Extension	8	18	10
Abduction	4	7	3
Adduction	5	20	15

As can be seen the mean increase of the non-cavitation group out-performed the cavitation group in all the actions. Abduction and Extension show large differences between the groups.

A manipulation without a cavitation can be considered a grade four mobilization (Haldeman, 1992). A grade four mobilization will cause separation of the joint surfaces within the zone of physiological movement of the joint (Leach, 1994:51). A grade four mobilization will cause the stimulation of the joint receptors in the joint being mobilized, as a result of the stretching and positional changes induced (Spencer, Hayes and Alexander, 1984). Stimulation of the joint receptors would alter the afferent innervation allowing a decrease in arthrogenic muscle inhibition (Suter, *et al.* 2000). Which would account for the increase of isokinetic strength values that have been observed in the non-cavitation group. However what is more difficult to explain is why the non-cavitation group out-performed the cavitation group.

Brodeur (1995) speculated that the sound of cavitation originated from the formation of a gas bubble within the joint, which is formed in response to the change of partial pressures within the joint caused by the separation of the joint surfaces during the action of a manipulation. The gas bubble is then absorbed back into the joint fluid over the next 20 minutes after the manipulation.

Based on the above theory of cavitation a gas bubble would be present within a manipulated joint up to 20 minutes after being adjusted. This gas bubble would be present in a joint that had an audible cavitation and not within a joint that did not have an audible cavitation (Brodeur, 1995).

Taking this into account could provide a rationale for the reason the non-cavitation group out-performed the cavitation group. The joint receptors play an important role in the formation, regulation and removal of arthrogenic muscle inhibition (Hopkins and Ingersoll, 2000). The presence of a gas bubble within the joint would stimulate the joint receptors (Spencer, Hayes and Alexander, 1984), sending information to the central nervous system that the joint was not in a normal state but in a refractive state (Brodeur, 1995). This information would perpetuate the presence of arthrogenic muscle inhibition and decrease the strength results that were obtained (Ingersoll, Palmieri and Hopkins, 2003). The gas bubble is however transient (Brodeur, 1995), so after the gas bubble has been reabsorbed the strength results could possibly increase to a level that is the same or higher than the non-cavitation group. The small, observed increase in strength of the cavitation group can be caused by the stimulation of some of the joint receptors during the manipulation, but the possible presence of a gas bubble would restrict the full effects of the manipulation.

A grade four mobilization would separate the joint surfaces causing stimulation of the joint receptors (Leach, 1994:17) reducing the effects of arthrogenic muscle inhibition (Suter, *et al.* 2000), without placing the joint in a refractive state. A grade four mobilization does not cause the presence of a gas bubble within the joint so there are no effects of the bubble stimulating the joint receptors perpetuating the effects of arthrogenic muscle inhibition. This could explain why the results of the non-cavitation group were higher than the cavitation group.

Investigation into the isokinetic strength values 15 - 30 minutes after the cavitation could add information in support of a gas bubble being responsible for

the lower cavitation groups' results. After 15 - 30 minutes the gas bubble would have been re-absorbed (Brodeur, 1995) ending the refractive phase and stopping the stimulation of the joint receptors, causing a decrease in arthrogenic muscle inhibition. If this holds true it would explain the difference between the cavitation and non-cavitation groups.

5.3.2.6. The effects of the side of involvement on the results

Out of a sample of thirty subjects, 12 presented with sacroiliac syndrome on the right and 18 on the left side.

For the following graphs
Series 1 (blue) = pre-manipulation
Series 2 (red) = post-manipulation
1 = **Right**
2 = **Left**

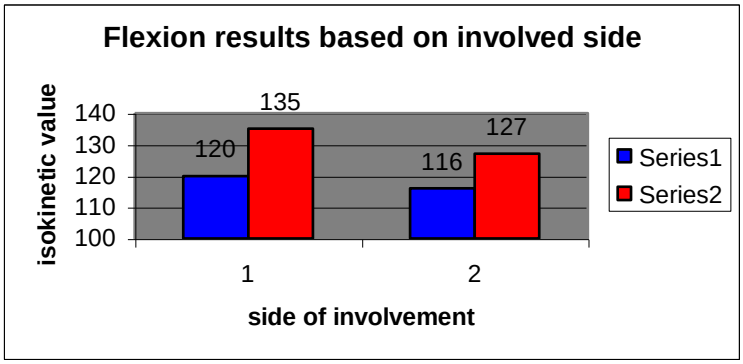


Figure 5.12. Flexion results based on involved side

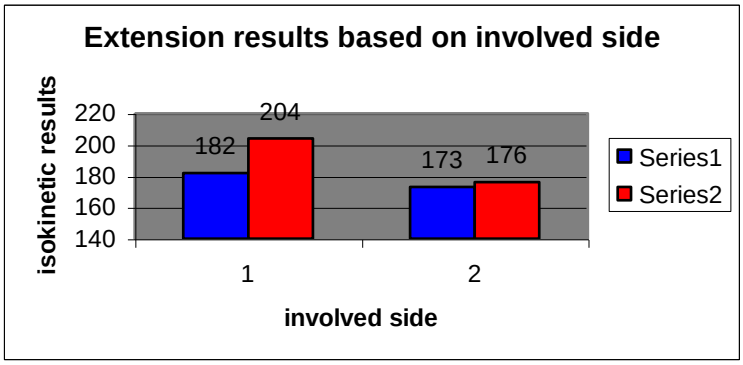


Figure 5.13 Extension results based on involved side

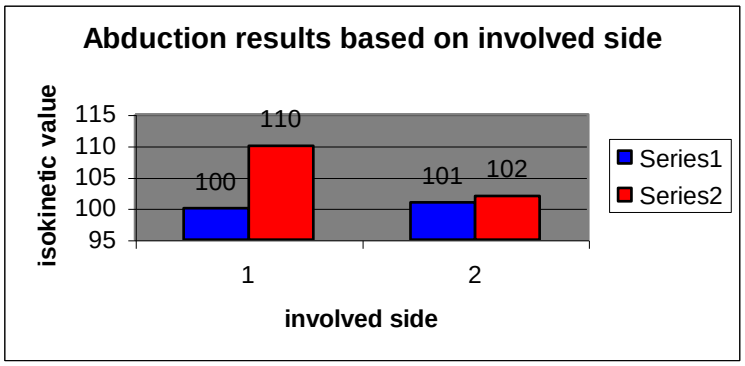


Figure 5.14. Abduction results based based on involved side

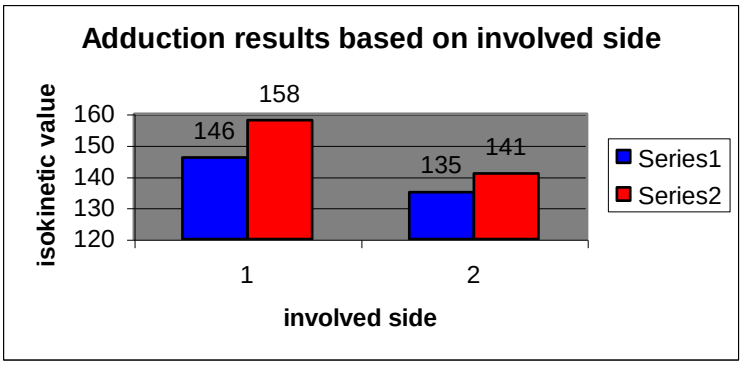


Figure 5.15 Adduction results based on cavitation

The table below shows the mean difference between the pre- and post-manipulation values for all the actions.

Table 5.3. Results based on cavitation

Action	Right	Left	Difference
Flexion	15	11	4
Extension	22	3	19
Abduction	10	1	9
Adduction	12	6	6

The right side showed a larger improvement than the left side for all the actions that were tested. Extension showed the largest improvement.

The reason for the larger improvement on the right side could be as a result of the researcher's involvement in the study. The researcher who manipulated the subjects is right hand dominant and more comfortable adjusting with the right hand. In the patient position for sacroiliac manipulation the right hand of the doctor is used to manipulate the left side of the patient.

In the sample, 18 subjects presented with involvement on the left side. Of the 18 manipulations 16 had cavitation, 2 did not. 12 subjects presented with sacroiliac involvement on the right side. 7of the 12 had cavitations when manipulated, 5 did not cavitate when manipulated. Therefore there were a higher number of cavitations amongst the subjects presenting with sacroiliac syndrome on the left side. This could be attributed to the doctor being right hand dominant and more comfortable adjusting the sacroiliac joint on the left side of the patient.

Although the left-hand side had more cavitations' than the right-hand side, the strength results on the left are lower than the right. The explanation for this difference is based on the same reason for the differences between the cavitation and non-cavitation group explained in 5.3.2.5.

The right hand side had fewer cavitations'. Therefore fewer results were influenced by the presence of a gas bubble, allowing higher isokinetic strength test results immediately after the manipulation.

CHAPTER SIX

6. RECOMMENDATIONS AND CONCLUSIONS

6.1. Introduction

This study aimed to investigate the immediate effects of an ipsilateral sacroiliac manipulation on hip muscle strength in patients suffering from a chronic sacroiliac syndrome. The sample consisted of thirty (30) male subjects.

The subjective measurements were taken using the numerical pain rating scale 101, these measurements were taken before and immediately after the manipulation.

Objective measurements for hip strength were taken using an isokinetic dynamometer (Orthotron II Isokinetic Rehabilitation System) for the actions of hip flexion, extension, abduction and adduction. These measurements were taken immediately before and immediately after an ipsilateral sacroiliac manipulation.

6.2. Conclusions

6.2.1. Subjective data

Analysis of the subjective data obtained from the numerical pain rating scale 101 revealed that the subjects experienced a reduction in their perception of pain due to the intervention of the sacroiliac manipulation.

This indicates that sacroiliac manipulation is an effective treatment for chronic sacroiliac syndrome.

From the findings of this study we were able to reject the null hypothesis and accept the alternate hypothesis that stated that an observed decrease in the subjects perception of pain was expected after an ipsilateral sacroiliac manipulation in patients suffering from a chronic sacroiliac syndrome.

6.2.2. Objective data

The objective isokinetic measurements evaluating hip strength for the actions of flexion, extension, abduction and adduction revealed that an ipsilateral sacroiliac manipulation was associated with immediate gains in hip muscle strength. These gains in hip muscle strength were as a result of an immediate decrease in arthrogenic muscle inhibition after the sacroiliac manipulation.

The gains in hip muscle strength followed a similar trend for all the hip actions tested. All the hip actions (flexion, extension, abduction and adduction) showed an increase in strength after the manipulation, there was not one action that significantly outperformed or responded differently to the manipulation.

These objective findings were in support of the hypothesis that sacroiliac manipulation caused an immediate effect on ipsilateral hip strength in patients suffering from chronic sacroiliac syndrome. The null hypothesis was therefore rejected and the alternate hypothesis was accepted for the objective data analysis of this study.

This study addressed the effects of

1. cavitation versus non-cavitation
2. side of involvement

on the strength results (discussed in chapter 5). This study speculated on the inferences drawn from these observations, however this study was unable to conclusively support these observations. Further investigation into these findings is recommended.

6.2.3. Arthrogenic muscle inhibition

The findings of this study lend support to the existence of arthrogenic muscle inhibition. The clinical effect of arthrogenic muscle inhibition explains the objective gains in muscle strength observed after the sacroiliac manipulation. Due to the increase in hip strength this study is able to support the application of manipulation in the treatment of arthrogenic muscle inhibition. Further speculation on the addition of sacroiliac manipulation into the rehabilitation protocol of the hip and in the treatment of arthrogenic muscle inhibition of the lower limb can be made.

6.3. Recommendations

Less financial constraints would have allowed the researcher to produce a more efficient and valuable study on the effects of arthrogenic muscle inhibition. In particular a bigger budget would allow a larger sample size that is always desired. Specialist services required for this study limited the sample size as the services were charged for. Thus the budget dictated the size of the study sample.

Arthrogenic muscle inhibition and isokinetic dynamometry are relatively new fields so consequently have limited available knowledge. This opens up these subjects to research. Manipulation is proving to be effective in reducing arthrogenic muscle inhibition, opening up this subject to further investigation.

The researcher can make a number of recommendations:

- Due to the anatomical and strength differences between the sexes the sample only included males. The exclusion of females was to overcome the differences between the sexes and add validity and homogenize the sample group. An investigation into the immediate effects of sacroiliac manipulation on hip strength in patients suffering from chronic sacroiliac syndrome should be conducted on a female sample to establish if the same conclusions from this study can be applied to the female population.

- This study investigated the **immediate** effects of sacroiliac manipulation on hip strength in patients suffering from chronic sacroiliac syndrome. Investigations into the longer term effects of sacroiliac manipulation on arthrogenic muscle inhibition should be conducted.

    a) an investigation into the strength levels 20 minutes after a manipulation (with cavitation) should be investigated. This

investigation would give valuable information into the explanation of the effects of manipulation based on the presence of a cavitation that were explained in chapter 5 (5.3.2.5.).

b) The short-term (24 hrs) and intermediate-term (>24 hrs) should be investigated to establish how long the effects of the manipulation last.

- This study looked at the effects of one intervention (manipulation). An investigation into the addition of a second manipulation at a follow-up consultation would be of benefit. This would help establish whether the effects of the manipulation are compounded to cause further increases in strength.

- Arthrogenic muscle inhibition can effect the contra-lateral limb. This study investigated the ipsilateral presence and effect of arthrogenic muscle inhibition, a study investigating the presence and effect of sacroiliac manipulation on the hip muscle strength of the contra-lateral limb would be beneficial and is recommended.

- Anecdotal evidence suggests that the gains in isokinetic hip strength shown by this study could be competitive with other treatment protocols for hip rehabilitation and inhibition. A comparative treatment trial between manipulation and other rehabilitation regimes and a combination of the two treatment protocols would be beneficial to gain practical information on the therapeutic benefits of manipulation with regard to muscle inhibition and rehabilitation.

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APPENDIX A

Case History

APPENDIX B

Revised Physical Examination

APPENDIX C

Lumbar Regional Examination

APPENDIX D

Letter of Information

Letter of information

Dear patient, welcome to this study.

Title of study:

The immediate effect of sacroiliac manipulation on hip muscle strength in patients suffering from chronic sacroiliac syndrome.

Supervisors: Dr M. Atkinson (031- 2042205)
Mr D. Jackson (031- 5662165)

Research student: Grant Matkovich (031- 2042205)

Institution: Durban Institute of Technology

Purpose of the study
Thirty patients suffering from low back pain (specifically chronic sacroiliac syndrome) will receive manipulative treatment for their lower back pain. The effects of the manipulation on hip muscle strength will be investigated to add to the knowledge on the effects of manipulation and the possible role manipulation may have in rehabilitation of the hip.

Procedures:
The first visit:
The initial consultation will take place at the Durban Institute of Technology Chiropractic Day Clinic. At this consultation patients will be screened for suitability to be included in the study. Suitability will be determined via a case history, physical examination and lower back regional examination. This appointment will take approximately an hour.

The second visit:
This appointment will occur at the Medigate Medical Centre in Umhlanga Rocks, and is subject to the availability of the biokineticist, Mr Jackson. The appointment will take place at these premises because they have the relevant facilities for isokinetic muscle testing. In the initial test, your hip strength will be tested with you performing four different hip actions. This is followed by a manipulative treatment. Immediately after the treatment your hip strength will be re-tested. This appointment will last approximately half an hour.

Directions to the Medigate Medical Centre are provided.

Risks/Discomfort:
The testing is relatively harmless, however some muscle stiffness after testing may be experienced.

Benefits:
The manipulative treatment that will be given is a common treatment intervention in the treatment for sacroiliac syndrome (low back pain). All appointments will be free of charge. On completion of your participation in this

study you are eligible for two free treatments at the Durban Institute of Technology Chiropractic Day Clinic.

New findings:
You have the right to be made aware of any new findings that are made.

Reasons why you can be withdrawn from the study without your consent:
- If you experience any discomfort during the isokinetic testing session.
- If you change any lifestyle habits, during your participation in this study, that may affect the outcome of this research. (eg. Change in medication, supplements or treatment)

You are free to withdraw from the study at any time, without giving a reason.

Remuneration:
You will **not** receive a travel allowance to get to the Medigate Medical Centre in Umhlanga.

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 with problems or questions:
Should you have any questions and want them answered by an independent source you can contact my supervisors 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.

Grant Matkovich	**Dr. M. Atkinson**	**Mr. D. Jackson**
(Chiropractic Intern)	(Supervisor)	(Supervisor)

APPENDIX E

Informed Consent Form

INFORMED CONSENT FORM

Date _____.

Title of research project:
The immediate effect of sacroiliac manipulation on hip muscle strength in patients suffering from chronic sacroiliac syndrome.

Name of supervisors: Dr. M. Atkinson (031- 2042205)
Mr. D. Jackson (031- 5662165)

Name of research student: Grant Matkovich (031- 2042205)

Name of institution: Durban Institute of Technology

Thirty patients suffering from low back pain (specifically chronic sacroiliac syndrome) will receive manipulative treatment for their low back pain. The effects of the manipulative treatment on hip muscle strength will be investigated.

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 effecting 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 question, please do not hesitate to contact my research supervisors, who will be able to assist you.

APPENDIX F

Numerical Pain Rating Scale 101

NUMERICAL PAIN RATING SCALE-101 QUESTIONNAIRE

Patient Name:_____. File No.:_____. Date:____.

Please indicate on the line below, the number between 0 and 100 that best describes the pain you experience when it is at its least. A zero (0) would mean “no pain at all”, and one hundred (100) would mean “pain as bad as it could be”. Please write only one number.

_____.

Please indicate on the line below, the number between 0 and 100 that best describes the pain you experience when it is at its worst. A zero (0) would mean “no pain at all”, and one hundred (100) would mean “pain as bad as it could be “. Please write only one number.

_____.

APPENDIX G

Advertisement

Do you suffer from

Low back pain

Are you **male** and between the ages of **18 – 45**?

If so then you may qualify for research being conducted at Durban Institute of Technology
CHIROPRACTIC DAY CLINIC

FREE TREATMENT

IS AVALIABLE ON COMPLETION OF THE STUDY

For more information contact
GRANT MATKOVICH
On (031) 2042205