A STUDY OF THE EFFECTIVENESS OF MYOFASCIAL TRIGGER POINT THERAPY ON ILIOTIBIAL BAND FRICTION SYNDROME IN SPORTS PERSONS

A Dissertation submitted in partial compliance with the requirements for a Master's Degree in Technology in the Department of Chiropractic at Technikon Natal.

BY
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I solemnly declare this is my own work in compilation and execution

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DEDICATION

This research is dedicated to my family, Alan, Chloë, and Lucy Saunders. To my parents Alan and Chloë, thank you for the emotional and financial support. Thank you for instigating my love for sport and thus my choice of career as a Chiropractor. I hope the enthusiasm you gave me rubs off on my work and thus the treatment results. Also, thank you for teaching me that by applying oneself and putting effort into something worthwhile, one can receive extreme self-satisfaction.

To my sister, Lucy, who I have always considered as a role model due to her excellent sporting talent and performances.

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To all you athletes, I hope the results of the study will ensure you are training and back on the road within a shorter period of time in future. Yet, let's hope you remember all the rules and prevent any further injury. I myself have suffered from Iliotibial Band Friction Syndrome which instigated this research dissertation.

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ABSTRACT

Iliotibial Band Friction Syndrome is a common problem for patients as well as physicians. Many treatments have been devised for Iliotibial Band Friction Syndrome, but there is very little research to substantiate which of these treatments is most effective. Myofascial trigger points are a frequently overlooked and misunderstood phenomenon in the medical curriculae, yet with correct diagnosis and appropriate treatment the prognosis of these trigger points is usually excellent. The aim of this placebo-controlled study was to justify the hypotheses which stated that myofascial trigger point therapy would be effective in the treatment of Iliotibial Band Friction Syndrome (IBFS), as compared to detuned ultrasound as a form of treatment.

Extensive advertising was undertaken to acquire subjects for the study. The subjects were examined for recognised signs and symptoms of IBFS.

The following criteria were taken into consideration before accepting patients into the study.

i) Patients suffering from any systemic disease were excluded from the study.

ii) The study excluded patients taking anti-inflammatories for their condition, yet, patients utilizing topical anti-inflammatories were not excluded.

iii) Patients with any neurological disease e.g.. lower motor neuron lesion were excluded from the study.

Patients were required to have the following cardinal signs and symptoms before being accepted in the study :

- pain over the lateral knee
- tenderness directly over the lateral femoral epicondyle, approximately 3cm proximal to joint line.

A sample size of thirty patients, which were taken from people residing in the Greater Durban Area were randomly divided into two groups of fifteen patients each. The experimental group received myofascial trigger point therapy of active trigger points in the tensor fasciae latae and iliotibial band. The control group received only detuned ultrasound over active trigger points and areas of pain. Myofascial trigger point therapy was delimited to dry needling, prescribed stretch exercises and patient education regarding perpetuating factors i.e. jogging uphill or downhill, increased pronated foot, genu varus, inadequate warming up and/or stretch exercises.

The following were also taken into consideration:

- Only active trigger points in the tensor fasciae latae and iliotibial band were treated.
- Biomechanical abnormalities, e.g. genu varum and high arched rigid feet were not considered or treated in this study.

The patient was treated for a maximum of ten treatments over a period of one month. A follow up consultation was scheduled one month later to evaluate the long term effects of treatment.

Subjective information regarding the patient's progress was collected by three questionnaires:
The Numerical Pain Rating Scale 101, McGill Pain Questionnaire and the Pain Disability Index. These were completed prior to the first, sixth and tenth treatment as well as at the follow-up consultation. Objective information was gathered with the aid of Wagner Force-

Dial algometer. Readings were taken prior to the first, sixth and tenth treatment and at the follow-up consultation.

The data was analyzed using Wilcoxon's paired singed rank test for intra-group analysis and Mann-Whitney U test for inter-group analysis. The statistical level of significance was set at 5% for both of these tests. Results were displayed with aid of tables.

When the results of both groups were analyzed it was found that the group receiving the myofascial trigger point therapy improved over the placebo group in both the subjective and objective measurements and in doing so, suggested all the stated hypotheses to be correct. It can thus be stated that myofascial trigger point therapy including stretching exercises and patient education, may be an effective means of treatment for IBFS.

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DEFINITION OF TERMS

ILIOTIBIAL BAND / ILIOTIBIAL TRACT

This is a thickened band of fascia on the lateral aspect of the thigh. Proximally the gluteus maximus and tensor fasciae latae re-inserted into it. Distally it is attached to the tibial tubercle (Gerdy's tubercle) the lateral femoral epicondyle and the linea aspera of the femur (Reid, 1992:424).

TENSOR FASCIAE LATAE MUSCLE

This muscle originates at the anterior part of the outer lip of the crest of the ilium and its function is to abduct and flex the thigh (Travell and Simons, 1983:217).

MYOFASCIAL PAIN SYNDROME

A pain syndrome characterised by pain in regional muscles and their fascia, accompanied by trigger points that refer pain and/or autonomic phenomena in a pattern specific for each muscle (Travell and Simons, 1983:3).

MYOFASCIAL TRIGGER POINT

Myofascial trigger points are areas of hyper-irritability in a muscle or its fascia that area described as hard indurated nodules that are specific with their reference to pain. Trigger points refer patterns of pain at rest or on motion that is specific to a certain muscle. An active trigger point is always tender, preventing lengthening of the muscle, weakening of the muscle and referring pain on direct pressure, mediating a "local twitch" response of the muscle when adequately stimulated. A latent trigger point is an area of hyperirritability in a muscle that does not produce spontaneous pain, and is only painful on palpation (Travell and Simons, 1983:3).

REFERRED PAIN

Pain that arises in a trigger point, but felt at a distance often entirely remote from its source. The pattern of pain is reproducibly related to its site of origin (Travell and Simons, 1983:3-4).

MYOFASCIAL TRIGGER POINT THERAPY

This includes :- dry needling, injection; spray and stretch; ischaemic compression; ice; exercise and patient education regarding : i) changes in lifestyle ii) occupational and recreational activities and iii) perpetuating factors (Travell and Simons, 1983:3-164).

For the purpose of study only dry needling; ice and stretch techniques will be used.

DRY NEEDLING

An invasive needling technique that involves specific and repeated insertion of an acupuncture needle into an active trigger point, using different angles of penetration of the needle whilst maintaining the original point of entry into the skin (Travell and Simons, 1983:3-164).

CRYOTHERAPY

Cryotherapy is the therapeutic use of cold (e.g. ice). It is useful in treatment of acute trauma, painful musculoskeletal conditions (e.g. IBFS) and neurological disabilities (Knight, 1985:9-193).

CHIROPRACTIC THERAPY

Any treatment that is performed by a person that is a qualified Chiropractor, more traditionally referring to the Chiropractic adjustment.

SUBJECTIVE CHANGES

These are the changes that are personally perceived by the patient i.e. how they feel with

regard to pain and disability.

OBJECTIVE CHANGES

These are the measurable changes in the patient's condition, as perceived by the practitioner. The changes are observed through the use of an algometer to assess sensitivity to pressure thereby determining irritability of the trigger point.

STRETCHING EXERCISE

In this study the stretching exercises refers to that specific stretch relevant to the tight tensor fasciae latae muscle and/or iliotibial band.

PATIENT EDUCATION

These are specific instructions that are given to the patient regarding his or her lifestyle and the perpetuating factors of the condition, with regard to preventing progression of the condition.

ALGOMETER

This is an instrument that reads pounds or kilograms per square centimetre and is useful in determining pain threshold (See Appendix 10A, 10B, 10C).

PLACEBO

This is any component of therapy or treatment that is deliberately or knowingly used without specific activity for the condition being treated. Note : for the purpose of this study the placebo treatment will be an application of detuned ultrasound over active trigger points.

CHAPTER ONE

INTRODUCTION

INTRODUCTION

Iliotibial Band Friction Syndrome (IBFS) is an overuse injury induced by friction between the bursa and the lateral femoral epicondyle, iliotibial band or underlying periosteum (Renne, 1975; Orava, 1978; Noble 1979; Noble et al. 1982 and Lindenberg et al. 1984). The injury is caused by friction of the iliotibial band over the lateral femoral epicondylar prominence (Renne 1975; Noble, 1979; Noble 1980; and Reid 1992:424) or by development of bursal tissue between these two structures with secondary inflammation (Lindenberg et al. 1984). Historically, IBFS has been considered a running related injury (Noble, 1979; Noble, 1980; Lindenberg et al. 1984; Baker, 1985 and Jones and James, 1987:280). This is due to the fact that during long distance running, repeated flexion and extension of the knee occurs and it is this movement that causes inflammation due to friction (Noble, 1980). This condition is frequently observed in skaters and cross country skiers (Orava, 1978; Noble, 1979 and Noble 1985:32); weight lifters (Orava, 1978); jumpers (Orava, 1978) and more recently, in cyclists, since the introduction of cleat pedals in 1985 (Noble 1985:32 and Holmes et al. 1993). Martens et al. (1989) state that in their study, IBFS was observed in football players.

IBFS accounts for one third to one fifth of all knee injuries (Noakes and Granger, 1990:96). Noakes (1992:452) states that, the IBFS "drags on endlessly" in certain runners. Noakes and Granger (1990:96) state that IBFS is the running injury which is most resistant to treatment, while Martens et al. (1989) concur reporting the condition to be a non self-limiting disease in athletes.

Most current authors are consistent in their approach to IBFS with regard to definition of the disorder, mechanism of dysfunction, signs and symptoms experienced and diagnostic procedure, but there tends to be less consistency when treatment of the problem is discussed. Treatment parameters must take into account the causative and perpetuating factors of IBFS, namely : training errors, inadequate shock absorption (Noble 1979; Noble, 1980; Sutker et al. 1981; Lindenberg et al. 1984 and Noakes, 1992:450-451) and a tight iliotibial band (Noble 1980; McBryde et al. 1985:410; Jones and James 1987:282; Reid, 1992:425 and Baker 1995). Treatment must also be aimed at decreasing the inflammation concerned with IBFS.

Many forms of treatment have been suggested for the disorder. These include ultrasound, electrical stimulation, iontophoresis, focal icing, whirlpool, laser treatment, assisted stretching, chiropractic manipulation, local heat application, massage, orthoses, prescribed anti-inflammatory medications, use of neoprene sleeves and hydrocortisone injections (Noble 1979; Noble 1980; Jones and James, 1987:283; Noakes 1992:451; Reid, 1992:425-427 and Baker 1995). Although all of the above methods conform to the principles of treatment, treatment success rate is very disappointing and IBFS is one of the most difficult injuries to treat (Noakes, 1992:452).

Thus it is important to establish a specific treatment regime for IBFS. Re-occurrence of the condition may also be prevented through education.

Travell and Simons (1983:219) state that a tight tensor fasciae latae as seen when tensor fasciae latae trigger points are present, contribute to iliotibial band tightness. According to Noble (1980), McBryde et al. (1985:41), Jones and James (1987:282), Reid (1992:425) and Baker (1995) an increased tightness in the iliotibial band causes an increased incidence of IBFS.

According to Travell and Simons (1983:19) and Fischer (1987), tender spots are not limited to muscles. They may be found in ligaments, joint capsules, periosteum, tendons and fascia, irrespective of the condition being caused by inflammation or injury (Travell and Simons, 1983:19 and Fischer, 1987). Thus, inflammation or injury to the iliotibial band, as in IBFS, causes tender points in the structure.

When the hip is actually flexed, the tensor fasciae latae pulls the iliotibial band anteriorly. When the hip is extended, the gluteus maximus shifts the band posteriorly (Lindenberg et al. 1984). Thus, in knee flexion greater than thirty degrees, the iliotibial band lies on or behind the lateral femoral epicondyle, whereas with the knee in the extended position, the iliotibial tract lies anterior to the lateral femoral epicondyle. Therefore, flexion and extension movements of the knee under stress can produce irritation and subsequent inflammatory reactions within the iliotibial tract, the underlying bursa and the periosteum of the lateral femoral epicondyle (Renne, 1975, Jones and James,

1987:280 and Reid 1992:424).

Thus, by treating trigger points in the tensor fasciae latae muscle and iliotibial band, it is hypothesized that the tightness of the iliotibial band will be decreased and that it will be of benefit to patients with IBFS.

Sandman (1981), Travell and Simons (1983:5), Bennett (1986), Simons (1988) and Baldry (1980:39), report that myofascial trigger points are amongst the most common cause of musculoskeletal pain seen in medical practice, yet, despite their frequency of occurrence, they are a poorly recognized and inadequately managed phenomenon. Myofascial pain may persist for decades (Gatterman, 1990:296), but Sandman (1981) states that with correct diagnosis and proper management of myofascial trigger points, prognosis is excellent. According to Sola (1991) trigger points may be considered as "weak points" within the muscle or fascia that are particularly sensitive to stress-induced changes.

Travell and Simons (1983:5); and Gatterman (1990:285) state that skeletal muscle accounts for forty percent or more of the total body weight and that frequency of occurrence of myofascial trigger points is not a surprising one. A possible cause of myofascial trigger points being overlooked when a diagnosis is made or a treatment protocol is devised, is due to the fact that little emphasis has been placed on the pathophysiology of muscles in both chiropractic and medical curriculae (Gatterman, 1990:285).

Myofascial Pain Syndromes are characterised by hypersensitive trigger points, described as hyperirritable loci within a taught band of skeletal muscle, located in the muscular tissue and/or its fascia. The spot is painful on compression and can induce referred pain and autonomic phenomena in a particular pattern in which they are located (Travell and Simons, 1983:12-15; Simons 1988; Baldry, 1989:43-47; Murphy, 1989 and Gatterman 1990:295). The Myofascial Pain Syndrome may present as a wide variety of clinical symptoms including pain, stiffness, reduced ranges of motion in joints, weakness, insomnia and autonomic dysfunction. The major complaint

on the part of the patient is pain (Sola and Williams, 1956; Simons, 1976; Mance et al. 1988).

Many forms of treatment have been suggested for Myofascial Trigger Point Therapy. The most common forms of therapy are : i) dry needling (Levine et al. 1976; Lewit, 1979; Mendelson et al. 1983; Baldry, 1989:36 and Garvey et al. 1989) ii) stretch and spray (Simons, 1976; Rubin, 1981; Sandman, 1981; Travell and Simons, 1983:27,63-65 and Mance et al. 1986) iii) ischaemic compression (Good, 1950; Good, 1951; Bonica, 1957; Simons, 1976; and Travell and Simons, 1983:75-76) iv) saline injections (Martin, 1952; Sola and Williams 1956; Frost et al. and Sola, 1984). Other forms of treatment for myofascial trigger point therapy include: ice rubs, moist heat, corticosteroids, analgesics, passive stretching, manipulation, deep stroking massage, ultrasound, biofeedback, high voltage stimulation, transcutaneous nerve stimulation and surgical removal of an extremely painful nodule (Yunus et al. 1981; Goldenberg, 1987; Murphy, 1989; Sola, 1991 and Haldeman 1992:523-524).

According to Bennett (1986) myofascial trigger points are not responsive to anti-inflammatory medications, including corticosteroids. Bennett (1986) states the symptomatic treatment with heat modalities, non-steroidal anti-inflammatory drugs, massage and local anaesthetic injections are of fleeting benefit, if at all.

The purpose of this investigation was to determine the effects of Myofascial Trigger Point Therapy (MFTPT) of the tensor fasciae latae and iliotibial band in patients presenting with IBFS, in terms of the patients subjective response as well as the measurable physical findings that may or may not occur in order to establish the efficacy of MFTPT as compared to the control group receiving merely detuned ultrasound. The objectives to be achieved included the measurement of alterations to both the subjective and objective presentation of patients in both the experimental and control groups with respect to pain and physical dysfunction. The measurements could then be statistically compared to determine the more effective treatment.

Needling of trigger points is a technique which is both easy to learn and use. With the results of this

study, practitioners treating IBFS will know whether MFTPT is of benefit to patients with this condition and this can only produce better results as a more effective treatment will result in a quicker recovery with greater long term effects.

CHAPTER TWO

REVIEW OF RELATED LITERATURE

2.1 INTRODUCTION

Iliotibial Band Friction Syndrome (IBFS) remains one of the most difficult injuries to treat (Noakes, 1992:452). According to Lindenberg et al. (1984), using conservative treatment, 83% (thirty of thirty-six) of their sample were cured completely of IBFS. Within three weeks, 58% (twenty-one of thirty-six) of the runners were completely cured. The remaining runners who were completely cured (nine of thirty-six), became symptom free within six weeks to six months. Of the six runners, whose treatment was not 100% effective, three had stopped running. The other three were re-evaluated and treated with in-shoe orthoses to limit subtalar joint pronation (as suggested by Subotnick). This is the reverse of what had initially been suggested. Of these three, two eventually became symptom free. The other runner reported that the orthoses had reduced his symptoms which enabled him to increase his training distance (Lindenberg et al. 1984).

According to Lindenberg et al. (1984) the mean recovery time to symptom free running was longer in runners who had symptoms for more than four weeks. However, there was no apparent relationship between the duration of symptoms and failure to respond to treatment, because two patients who were not 100% relieved had symptoms for less than two weeks.

Travell and Simons (1983:18) state that frequently the response to specific myofascial therapy is immediate disappearance of spot tenderness, referred pain and local twitch response, with release of the muscle's restricted motion. The tension of the palpable band is less likely to disappear immediately when myofascial syndrome has been present for a long period of time, i.e. months or years.

2.2 AETIOLOGY OF ILIOTIBIAL BAND FRICTION SYNDROME

Renne (1975); Lindenberg et al. (1984); Jones and James (1987:280) and Reid (1992:424) state that friction is caused by flexion and extension movements of the knee, bringing the thickest portion of the band, which is adjacent to the lateral femoral

epicondyle, anterior to the axis of motion in the last thirty degrees of extension. The band moves posterior to the axis of knee motion and condylar prominence during flexion movement past the thirty degree point, i.e. when the knee is straight, the fascia lies in front of the lateral femoral epicondyle, but as the knee bends, the fascia moves towards that bony point. When the knee has bent through greater than thirty degrees, the fascia may touch the femoral epicondyle, especially if the iliotibial band is tight, and its contact is believed to cause pain in this syndrome. Pena (1991) states that in the sport of cycling, flexion and extension occur approximately four thousand eight hundred times an hour, at an average pedalling cadence of eighty revolutions per minute. The iliotibial band is thus especially susceptible to irritation in cycling.

Another point of friction is at the greater trochanter due to external rotation of the hip during running, skating and cross-county skiing. This can lead to the potential for trochanteric bursitis (Reid, 1992:424).

Noakes (1992:450) states that there is no consensus to the cause of IBFS, yet the following causes have been isolated:

2.2.1 A Tight Iliotibial Band

McBryde et al. (1985:410), Jones and James (1987:282); Martens et al. (1989); Reid (1992:425) and Baker (1995) state that genu varum and leg length discrepancies cause tightness of the iliotibial band which predisposes IBFS. Jones and James (1987:282) stated that leg length discrepancy cause a lateral pelvic tilt with subsequent stretching of the tract over the lateral femoral epicondyle of the lower leg, thus contributing to IBFS.

According to Lindenberg et al. (1984); in their study, 56 % of runners had leg length discrepancies. However, the side of the discrepancy did not correlate with the side of the injury. Lindenberg et al. (1984) therefore concluded that leg length

discrepancies may not contribute to IBFS.

According to Noble (1980) tightness of the iliotibial band may be structural or functional. Yet, structural tightness is extremely difficult to assess. Iliotibial tract contractures are seen after certain paralytic disorders. However, these contractures are not seen in athletes (Noble, 1980). Noble (1980) quotes Subotnick who states that excessive internal rotation of the tibia on the femur also causes increased tension on the iliotibial band. This may be found on excessive in-toeing when running or in abnormal pronation of the foot during the stance phase of running.

According to Noble (1979), genu varum predisposes a runner to IBFS by increasing the tautness of the band over the lateral femoral epicondyle and thereby increasing the likelihood of friction between these structures, particularly if there is a prominent lateral femoral epicondyle. Sutker et al. (1981) and Sutker et al. (1985) state that a cavus foot results in a more pronounced varus stress on the knee and thus results in IBFS.

The injury occurs on the side of the body corresponding to the side of the road on which the runner runs (Noble 1979; Lindenberg et al. 1984; Noakes, 1984; Firer, 1989; Noakes, 1992:451 and Reid 1992:424) i.e.. runners who ran on the right side of the road usually develop the injury on the right hand side. According to Lindenberg et al. (1984), 70% of his twenty three runners using the right side of the road developed right-sided injuries and the only runner using the left side developed a left sided injury. Therefore, 71% of runners who favoured the left or right side of the road sustained injuries to the leg on the down-sloping side of the cambre. According to Firer (1989) in his study, right-sided IBFS was dominant. He states that this is due to the cambre of the road. He explains that in South Africa (where his study was performed), right hand drive occurs and thus one

drives on the left side of road. For safety reasons, runners face the oncoming traffic and therefore develop right-sided IBFS.

2.2.2 Inadequate Shock Absorption

Jones and James (1987:282) and Noakes and Granger (1990:99) considered inadequate shock absorption as a cause of IBFS. Baker (1995) states that shoe changes and wear lead to inadequate shock absorption and thus predispose IBFS. According to Sutker et al. (1981); Sutker et al. (1985); Noakes and Granger (1990:99) and Noakes (1992:450) high arched, rigid, cavus feet are unable to absorb the shock adequately, which is transferred to the iliotibial band, and thereby results in injury. Yet, on the other hand, some runners with extremely flat feet, which are also unable to absorb shock adequately, may be equally at risk (Noakes and Granger, 1990:99). Sutker et al. (1981) states that running on hard surfaces can predispose IBFS. Hard shoes with poor shock absorbing qualities also increase the incidence of IBFS (Noakes, 1992:450).

There is some controversy as to whether inadequate or excessive subtalar joint pronation is a perpetuating factor of IBFS. Evidence for decreased ankle pronation includes a study performed by Noakes (1992:450) in which approximately seventy percent of cases with IBFS occur in runners with decreased ankle pronation. He also states that this decreased ankle pronation results in lower limb failure to absorb shock adequately and thus lead to IBFS. In the same way, orthotics that restrict ankle pronation may lead to IBFS.

Further evidence for decreased ankle pronation as a cause or perpetuating factor of IBFS include that this injury occurs predominantly in the leg closest to the down side of the cambre and it is running in this position that restricts pronation (Lindenberg et al. 1984). They continue and state that inadequate pronation may be a contributing factor to IBFS due to the fact that during treatment, patients with

this injury respond rapidly to soft running shoes which control pronation poorly. This evidence for decreased ankle pronation is consistent with the recommendations of Noble et al. (1982) who suggest that lateral heel wedges should be prescribed, especially for patients with a tight iliotibial band.

Excessive subtalar joint pronation may be a predisposing factor to IBFS. Jones and James (1987:282) state that the tibia internally rotates with pronation of the foot. If the internal rotation is increased and prolonged with excessive pronation, then more transverse rotation must be absorbed by the knee joint. With this excessive and prolonged internal rotation, the insertion site of the iliotibial band is drawn anteromedially, therefore drawing the iliotibial tract tighter across the lateral femoral epicondyle, produces excessive irritation to the tract and the underlying bursa and periosteum (Jones and James, 1987:282).

Lindenberg et al. (1984) state that 17% (six of thirty-six) of a sample of patients with IBFS were unable to be fully cured. The individuals who continued running were ultimately helped when they were prescribed orthotics to limit ankle pronation, the reverse of what had initially been suggested by Lindenberg et al. (1984).

Evidence in support of excessive subtalar joint pronation as a predisposing factor of IBFS include that 33 % (twelve of thirty-six) of runners of the Lindenberg et al. (1984) sample were running in soft running shoes (with poor pronation control) at the time of treatment. Lindenberg et al. (1984) stated that 72 % (twenty-six of thirty-six) of his sample group had previous running injuries visually associated with excessive ankle pronation. These included: Runner's Knee; Shinsplints and Achilles Tendonitis.

Thus, according to Lindenberg et al. (1984) both excessive and inadequate ankle

joint pronation can contribute to IBFS.

2.2.3 Training Errors

Some of the more common causes of IBFS include the following :

2.2.3.1 Heavy Training Mileages

According to Noakes and Granger (1990:97), because increased mileage regimes predispose IBFS, this injury occurs especially in peak training season. In South Africa, the injury may be seen in epidemic proportions in March/April each year as runners increase their training immediately before the Comrades Marathon.

2.2.3.2 Alterations in Terrain

According to the contributing authors, excessive hill work, especially downhill running, running on a hard and/or cambred surface predisposes IBFS. Noble (1980) found that 22% of runners with IBFS developed symptoms due to excessive hill training. Holmes et al.(1993) stated that symptom onset correlated with increased hill training in 14% of cyclists.

According to Travell and Simons (1983:221-222), chronic overload, in the form of the training errors mentioned, predisposes towards the development of tensor fasciae latae trigger points.

2.2.3.3 Other Training Errors

Sudden increases in training; too much racing; an increase in speed or interval training and inadequate warming up may be a cause of IBFS (Noble, 1979; Noble, 1980; Jones and James, 1987: 281; Noakes, 1992; 450-451 and Reid, 1992; 452).

2.2.4 Other Factors Causing Iliotibial Band Friction Syndrome

Baker (1995) states that talar fixation was the causative factor in iliotibial band tendinitis in a specific case study. She noted that patients presented with pain over the right lateral knee approximately twenty four hours after an eversion sprain of the right ankle. Talar fixation as a cause of iliotibial band tendonitis is explained because subtalar motion and tibial rotation are interdependent in the weight bearing foot and with abnormal joint motion of the subtalar and knee joints, muscular and ligamentous stress is also increased (Baker, 1995).

Holmes et al. (1993) states that cyclists that exhibit external tibial rotation of greater than twenty degrees when placed into a straight head of slightly toe-in cleat position, place significant stress on the distal iliotibial band as it crosses the lateral femoral epicondyle and it is thus considered a potentially contributing factor in the development of IBFS. Stress on the iliotibial band can also result from riding with the cleats too far internally rotated. Saddle height, which determines the degrees of knee flexion and extension with each pedalled stroke, can also be a contributing factor. A saddle that is too high results in knee extension beyond one hundred and fifty degrees causing the distal iliotibial band to abrade across the lateral femoral epicondyle. Saddles that are too far back cause the cyclists to reach for the pedal with resultant stretch on the iliotibial band (Holmes et al. 1993).

According to Noble (1980) and Jones and James (1987:282), excessive prominence of the lateral femoral epicondyle increases the friction. Renne (1975) found no radiological evidence of increased size or prominence in his patients compared to normal controls.

Sutker et al. (1985) found runners with apparently normal feet, developed IBFS after switching to shoes with a so - called "varus stretch". While these shoes may be excellent for a runner with pronated feet, the increased lateral knee stress can

initiate the syndrome.

2.3 PREVALENCE AND INCIDENCE OF ILIOTIBIAL BAND FRICTION SYNDROME

Noakes and Granger (1990:96) states that this injury accounts for one third to one fifth of all knee injuries in distance runners. Noble (1980) states that either knee can be affected, yet rarely are both knees affected.

Lindenberg et al. (1984) found that runners most susceptible to IBFS have been running for four years or less, have run at least one standard marathon and train more than forty kilometres per week. These findings are remarkably similar to those of Orava (1978); Noble (1979) and Sutker (1981). They also run with the affected leg on the lower side of a cambered road and are more likely to be running on a hard shoe. They have normal or varus knee alignments, varus rearfoot and forefoot and leg length discrepancy that does not relate to the side of the injury (Lindenberg, et al.1984).

According to Reid (1992:425), IBFS occurs in any age group. Yet, Lindenberg et al. (1984) found that runners most commonly affected with IBFS were between the ages of twenty and forty years old. In agreement, Sutker et al. (1981) and Sutker et al. (1985) found that the average age in their study was thirty one years old.

Sutker et al. (1981) and Sutker et al. (1985) reported that in their study 75% were male. Lindenberg et al. (1984) reported in their study, of thirty six patients, thirty being men and six being women. Lindenberg et al. (1984) stated that this predominance of males may be to the fact that a greater number of males participate in running activities and men usually run further distances. Also, women usually have a less distinct iliotibial band, greater valgus at the knee, more ligamentous laxity, a less prominent lateral femoral epicondyle and more subcutaneous fat that men. All these factors make women less likely to develop irritation under the iliotibial band. The iliotibial band does not appear to be as taught in flexion in valgus as in neutral or varus alignment (Sutker et al. 1981; Sutker et al. 1985 and

Reid, 1992:425).

Sutker et al. (1981) and Sutker et al. (1985) state that in their study, IBFS was rarely seen in endomorphs, which is probably due to the fact that they do not run as far. In addition, fat in the tissue around the knee may serve as lubrication under the taught iliotibial band (Sutker et al. 1981 and Sutker et al. 1985).

According to Noble (1980), this syndrome has a higher incidence in areas where long distance running is in vogue, such as South Africa or where the climate is cool and running surfaces are slippery.

Weiss (1985) noted four cyclists amongst a group of one hundred and thirty two participants on an eight-day bicycle tour who presented with iliotibial symptoms. Martens et al.(1989) reported that two cyclists amongst a group of nineteen were treated surgically for IBFS. Holmes et al.(1993) quotes Burke who hypothesizes that the number of cyclists bothered with IBFS has increased since the introduction of the rigid clipless pedal system in 1985.

2.4 PREVALENCE OF MYOFASCIAL TRIGGER POINTS

According to Travell and Simons (1983:5), myofascial trigger points are extremely common. Individuals of any age or sex can develop trigger points (Travell and Simons, 1983:13). Latent trigger points are more common than active trigger points (Travell and Simons, 1983:5). No literature was found stating the prevalence of myofascial trigger points in IBFS.

Trigger points are not only found in muscle tissue alone. In addition, they are found in fascia, tendons, joint capsules and ligaments. (Travell and Simons, 1983: 19). Fascial trigger points, as in trigger points in the iliotibial band, give sharply localized pain, whilst trigger points in muscle give diffuse pain (Kellgren, 1938). Pain arising from both fascial

and muscular trigger points may be referred (Kellgren, 1938; Hockaday and Whitty 1967).

2.5 PERPETUATING FACTORS OF TENSOR FASCIAE LATAE TRIGGER POINTS

Simons (1976); Travell and Simons (1983:103-155); Graff-Radford et al. (1987), Baldry (1989:49) and Gatterman (1990:287) highlight some of the perpetuating factors of myofascial trigger points. According to Melnick (1954) and Sandman (1981) trigger points may spontaneously subside once perpetuating factors have been removed. Thus, in any treatment of myofascial pain these factors must be taken into consideration and corrected or eliminated to ensure effective treatment of the condition.

Mechanical stresses, for example, skeletal asymmetry, and muscle abuse may lead to myofascial trigger point formation (Travell and Simons, 1983:103-155). Murphy (1989) states that trigger points may be formed due to prolonged muscle spasm, direct or indirect injuries or orthopaedic abnormalities. In the case of tensor fasciae latae trigger points, perpetuating factors include excessive hill running, especially without appropriate foot support, as in a Morton's foot structure (a long second toe) or should excessive pronation of the foot occur. Inadequate shock absorption due to rigid cavus feet and running on hard surfaces, as well as sudden increase in running mileage and inadequate warming up, are other factors that predispose to the development of tensor fasciae latae trigger points (Travell and Simons, 1983:221-222). Sola and Williams (1956) and Baldry (1989:40), state that the most common predisposing factor to myofascial trigger point formation is mechanical stress.

Deficiency in elements that are required for normal muscle metabolism may lead to the development of myofascial trigger points. The elements include: Vitamins B1, B6, B12 and C as well as folic acid, calcium, potassium and iron (Travell and Simons, 1983:103-155 and Gatterman, 1990:287)).

Endocrine and metabolic inadequacies which perpetuate trigger point formation include:

hypometabolism due to hypothyroidism, hyperuricaemia and hypoglycemia. Any condition that impairs muscle metabolism, such as anaemia or hypoxia likely leads to myofascial trigger point formation (Travell and Simons, 1983:103-155 and Gatterman, 1990:287).

Chronic infection is also believed to be a predisposing factor of trigger points (Travell and Simons, 1983:103-155 and Gatterman, 1990:287).

Other perpetuating factors include : allergies, fatigue and cold, damp weather, excessive heat, prolonged bed rest or immobility (Travell and Simons, 1983:103-155) and Gatterman, 1990:287.)

Murphy (1989) states that without correction of the perpetuating factors, it may be impossible to resolve the patient's problem. In some patients, elimination of these factors results in complete relief without any further treatment of the muscles.

2.6 AETIOLOGY AND DEVELOPMENT OF MYOFASCIAL TRIGGER POINTS

Trauma and microtrauma to the muscle result in Myofascial Pain Dysfunction Syndrome (MPDS) as it biologically initiates pain (Good, 1951; Yunus 1981; Mance et al. 1986; Baldry, 1989:40 and Gatterman, 1990:29). Travell and Simons (1983:221) state that tensor fasciae latae trigger points may be due to sudden trauma. This may be in the form of direct injury to the muscle, or a sudden strain, or by it being subjected to excessive or unusual exercise (Awad, 1973 and Baldry, 1989:40). Thus, in the case of IBFS, trigger points may develop in the iliotibial band and tensor fasciae latae muscle when the patient increases running mileage. According to Baldry (1989:40), the activation of trigger points may occur more gradually such as when muscles are subjected to repeated episodes of minor trauma or are repeatedly overloaded.

According to Awad (1973) and Mance et al. (1986), in this pathological process, an increased concentration of acid mucopolysaccharides, water, chloride and mast cells

accumulate in the area. Sandman (1981) supports this concept by stating that it is generally accepted that an organic pathological process occurs at the point of sustained contracture, along with a decrease in blood supply and accumulation of metabolites.

Baldry (1989:40) cites trauma inflicted on the muscle by direct injury, sudden strain or excessive or unusual exercise as the main cause of trigger point development.

Awad (1973); Travell and Simons (1983:32-37); Baldry (1989:41); Murphy (1989) and Gatterman (1990:291) propose the following to explain the development to trigger points. Many aetiological factors interact to create myofascial trigger points by the following process. Trauma to the muscle from acute or chronic strain results in tissue damage. This often includes disruption of the sarcoplasmic reticulum which results in the release of stored calcium and the inability of the damaged sarcoplasmic reticulum to remove it from the site of injury. The chronic stress of the resultant sustained contraction, or excessive fatigue during repeated contractions, may cause a vulnerable region of the muscle to become disproportionately strained, repeating this same process. The availability of extra calcium (in the presence of normal amounts of ATP) to myofibrils results in sustained contraction of the muscle fibre. This produces a palpable taught band in the muscle.

According to Baldry (1989:41) and Gatterman (1990:291), local tenderness and referred pain occur when nerve endings in the area of trauma are sensitised by accumulation of substances produced by the following mechanisms:

- small blood vessels are disrupted and release platelets which in turn release serotonin
- mast cells containing histamine are damaged when connective tissue is disrupted, resulting in the release of histamine
- metabolites such as prostaglandins aggregate due to the sustained contraction impeding blood flow to the area.

The reduced local blood flow resulting from the sustained contractions is compounded by vasoconstriction that is activated because of trigger point sensory - fibre input to the central nervous system. Further consequences of sustained contraction are that it creates a region of uncontrolled metabolism that can result in additional mast cell liberation of histamine and subsequent depletion of local ATP. A progressive failure of relaxation eventually results in muscle contracture due to the energy (required from splitting ATP) not being available to "recock" the contractile mechanism (Gatterman, 1990:291).

Thus, a self-perpetuating local muscular condition is created which is painful, resists stretching and results in decreased range of motion and generalized disability (Gatterman, 1990:291). Normal function may be restored by stretching all of the involved muscle fibres far enough apart to eliminate contraction. Only then can enough ATP accumulate to restore the sarcoplasmic reticulum to normal. Once this has happened the renewed circulation can begin to slowly remove the build up of metabolites (Gatterman, 1990:291).

2.7 PATHOLOGY OF ILIOTIBIAL BAND FRICTION SYNDROME

Orava (1978) noted a reddish brown bursal thickening under the iliotibial band adjacent to the prominence of the lateral femoral epicondyle. Renne (1975) noted that three millilitres of clear, yellow viscous fluid was aspirated from the involved bursa in a patient with IBFS. Martens et al.(1989) states that during surgical procedures to the Iliotibial Band, macroscopic inflammation of the tissue is encountered over and around the lateral femoral epicondyle. Histological examination of this tissue revealed fibrous tissue with a synovial - like structure showing many areas of mucoid degeneration or fibrinoid necrosis (Noble 1979; Noble 1980 and Martens et al. 1989).

2.8 PATHOLOGY OF MYOFASCIAL TRIGGER POINTS

Yunus (1981) and Travell and Simons (1983:6-7) quote Gowers, Llewellyn and Jones who state that trigger points are due to inflammation and hyperplasia of connective tissue. But this was not substantiated in most subsequent biopsy studies. Travell and Simons (1983:7) quote Schade who later postulated that trigger point formation was due to an increase in the viscosity of the muscle colloid. German biopsy studies showed characteristic non-specific changes by light microscopy (Travell and Simons, 1983:7). Simons (1976), Travell and Simons (1983:7-8) and Baldry (1988:43), quotes Awad, Fassbender and Wegener who reported ultramicroscopic findings in biopsies of muscles showing abnormality of the contractile elements in the muscle, therefore indicating myofascial trigger points. Awad (1973) observed abnormally lengthened sarcomeres, large clusters of blood platelets and mast cells discharging their granules. He inferred, from this, that trauma is likely to be an initiator of a disease process that would produce degranulation of mast cells both directly and indirectly. Indirectly, platelets extravasated by bleeding, due to trauma, release serotonin which may produce prolonged vasoconstriction that increases oedema (which in itself aggravates vasoconstriction). Finally, the oedema also triggers degranulation of mast cells. Fassbender described various changes in sarcomeres ranging from partial damage, to gross disruption but still recognisable, to them being completely destroyed and leaving nothing but a fine granular residue within the sarcomere. He inferred that the primary cause of myofascial trigger points is hypoxia which leads to loss of mitochondria function and therefore loss of oxidative metabolic pathways in the muscle. This results in destruction of the sarcomere. The findings were not interpreted as an inflammatory process (Simons, 1976; Travell and Simons, 1983:7-8 and Baldry, 1989:43).

Simons (1976) and Baldry (1989:43) quote Brendstrup who found that fibrositic muscle showed increased concentration of acid mucopolysaccharides, and increased water content and chloride content. Brendstrup found that the firmer feel to the area of the muscle was due to oedema. Good (1950) theorizes that the condition is a result of disturbed circulation.

2.9 CLINICAL PRESENTATION OF IBFS

2.9.1 Symptoms of Iliotibial Band Friction Syndrome

The patient presents with severe stabbing pain (Holmes et al. 1993) directly over the lateral femoral epicondyle, approximately three centimetres proximal to the joint and related to activity (Renne 1975; Noble 1979; Lindenberg et al. 1984; Jones and James, 1987 : 280-281; Henderson, 1989; Noakes 1992:449; and Baker 1995). According to Lindenberg et al. (1984); Noakes and Granger (1990: 97) and Noakes (1992,449), the athlete is able to play other sports, for example squash, rugby and tennis, without discomfort. IBFS is usually seen in sports that require continuous running (Orava 1978; Noble 1979; Noble 1980; Sutker et al. 1981; Sutker et al. 1985 and Reid 1992). The pain which increases with mileage is initially of a mild aching type (Reid, 1992:424).

According to Noble (1980), Lindenberg et al.(1984), Noakes and Granger (1990:97), and Noakes (1992:449), the pain once present, prevents further running. Yet, Noakes (1992:449), states that the pain subsides immediately when the athlete stops running (i.e. the pain is absent at rest) but should he commence running again, the pain returns rapidly. According to Noble (1980) and Reid (1992:424), if the athlete persists in pushing themselves the pain continues, even when walking and ultimately persists between training sessions. In mild cases, pain may improve after the runner has warmed up (Noble, 1980 and Jones and James 1987:280-281). In severe cases, even prolonged rest does not settle the pain (Noble, 1980). Pain comes on at a remarkably constant point in their work out (Reid 1992 : 424). During running, pain usually becomes so severe that it limits running distance, varying from runner to runner, between one hundred metres and sixteen kilometers (Sutker et al. 1981; Sutker et al. 1985, Noakes and Granger 1990:97 and Noakes, 1992:449). Physical examination reveals point tenderness and pain with provocative testing (Orava 1978; Noble, 1979; Noble et al. 1982; Jones and James 1987:280-281 and Noakes and Granger, 1990:97. Other symptoms of IBFS include lateral hip pain (Baker, 1995), soft tissue swelling over the lateral femoral epicondyle and "wet

leather crepitus" (Noble, 1980).

Pain associated with IBFS sometimes radiates distally to the tibial attachments, calf proximally to the lateral thigh (Orava, 1978; Noble, 1980; Jones and James, 1987: 280-281 and Reid, 1992:424). Walking downstairs (Lindenberg et al. 1984; Noakes and Granger, 1990:97 and Reid 1992:424) or downhill running (Noble 1979; Noble 1980; Jones and James, 1987:280-281; Noakes and Granger, 1990:97 and Noakes, 1992:449) or excessive striding (Noble, 1980) especially aggravate the symptoms. Increased length of stride while running will also cause excessive compression between the iliotibial band and the lateral femoral epicondyle (Noble, 1980). Increased compression may be caused by lateral heel wear in the running shoe (Noble, 1980). Walking with the knees extended or "stiff legged" may provide relief (Noble 1979; Noble 1980; Jones and James 1987:280-281; Henderson 1989 and Reid, 1992:425) because the iliotibial band remains anterior to the epicondyle with the knees in extension (Renne, 1975 and Henderson, 1989). Severity of IBFS can be graded on a 1-5 scale (See Appendix 13).

2.9.2 Signs of Iliotibial Band Friction Syndrome

The most important clinical finding is an area of exquisite tenderness found on palpation over the lateral femoral epicondyle and lower third of the iliotibial band (Noble 1979; Sutker et al. 1981; Noble 1985:32; Sutker et al. 1985; Noakes, 1992:449 and Holmes et al. 1993). Swelling over the lateral femoral epicondyle may be present in severe cases (Noble, 1985: 32).

2.10 CLINICAL PRESENTATION OF TENSOR FASCIAE LATAE TRIGGER POINTS

2.10.1 Symptoms of Tensor Fasciae Latae Trigger Points

The pain is referred from trigger points in specific patterns characteristic of each muscle (Travell and Simons, 1983:13; Mance et al. 1986; Graff-Radford et al. 1987; Simons, 1988; Yunus et al., 1988; Baldry, 1989:47; Gatterman, 1990:295

and Sola 1991). Melnick (1954) and Travell and Simons (1983:13) state that symptoms are induced whenever the trigger point is irritated. Pain does not precisely follow dermatomes, myotomes or sclerotomes and it may involve many segments. (Good, 1951; Sola, 1981 and Travell and Simons, 1983:14). Tensor fasciae latae trigger points refer pain down the lateral pelvis and anterolateral thigh including the greater trochanter and the lateral knee as far as the midcalf (Sola and Williams, 1956; Sola, 1984 and Sola, 1991).

According to Sola and Williams (1956) and Hockaday and Whitty (1967) the mechanisms that underly referred sensations are uncertain. Yet, according to Travell and Simons (1983:31-33), four mechanisms are known to explain referred pain: 1) transmission by autonomic pathways; 2) peripheral branching of pain nerves; 3) convergence - projection and 4) convergence facilitation. The latter occurs within the central nervous system. Travell and Simons (1983:16) state that deep tenderness and dysthesiae are commonly referred to the zone of referred pain.

The pain is described as deep, dull and aching and pain intensities range from discomfort to severe (Sola and Williams, 1956; Bonica, 1957; Travell and Simons, 1983:6-13 and Gatterman, 1990:295).

The patient presents with a history of chronic overuse, for example, excessive hill running as well as inadequate warming up and stretching. Trigger points are activated directly by acute overload; overwork fatigue; direct trauma and chilling.

Trigger points also cause stiffness and weakness of the involved muscles (Yunus et al. 1981; Travell and Simons, 1983:15; Goldenberg 1987; Baldry, 1989:43 and Gatterman, 1990:295;). According to Goldenberg (1987), the stiffness and pain

are of an aching character and the pain is worsened by overuse and straining. A local muscle spasm is also present.

2.10.2 Signs of Tensor Fasciae Latae Trigger Points

The trigger point is found in a hard indurated band and is exquisitely tender to palpation (Awad, 1973; Sandman, 1981; Travell and Simons, 1983:6; Simons, 1988 and Gatterman, 1990:295). Muscles adjacent to trigger points may also feel tense on palpation (Travell and Simons, 1983:16).

Simons (1976) and Mance et al. (1986) state that there are three types of hardening felt on palpation of trigger point:- nodular, spindle - shaped and rope like. Pressure over this area produces pain felt in the zone of reference. Travell and Simons (1983: 16-17) state that deep tenderness is commonly referred by the trigger point to the zone of referred pain. Disturbances of non-sensory function are sometimes included in the pain referral zone.

According to Travell and Simons (1983:16); Baldry (1989:47); and Gatterman (1990:2), digital pressure applied to an active trigger point may elicit a "jump sign" i.e. the patient may jump, cry out and recoil from the pain. The patients response will be greatly exaggerated in proportion to actual amount of pressure. The jump sign is frequently seen in response to digital pressure on taught, flat muscle surfaces, for example the tensor fasciae latae (Murphy, 1989). A local twitch response may be elicited when the trigger point is rolled under the fingers. This is a transient contraction of the muscle that can be felt by the examiner. The twitch may be vigorous enough to cause a perceptible jerk of the body part (Travell and Simons, 1983:16; Simons, 1988; Baldry, 1989,47; Murphy, 1989; and Gatterman, 1990:296).

According to Bonica (1957), Simons (1976), Sola (1984) and Mance et al. (1986)

the muscle exhibits protective spasm which causes reduced ranges of motion. MacDonald (1980) states that when active trigger points are present, passive or active stretching of the affected muscle increases the pain. Pain is increased when the affected muscle is strongly contracted against fixed resistance. This effect of a trigger point is most marked when the muscle is placed in a shortened position prior to the contraction effort (Simons, 1976; Travell and Simons, 1983:16; Mance et al. 1986; Simons, 1988 and Gatterman, 1990:296). When the tensor fasciae latae muscle is tight, adduction is limited to less than fifteen degrees. The tight tensor fasciae latae therefore contributes to the tight iliotibial band, therefore increasing the incidence of IBFS. The increased tension of the taught bands will not permit the tensor fasciae latae or iliotibial band to extend to its full range. (Travell and Simons, 1983:16).

Travell and Simons (1983:16) state that the maximum contractile force of the affected muscle is weakened. The weakness of the tensor fasciae latae can usually be demonstrated by testing the resisted isometric movements.

According to Travell and Simons (1983:17) and Yunus et al.(1988) routine laboratory tests show no abnormalities or significant changes attributable to myofascial trigger points. But, thermograms of skin overlying active trigger points show areas of increased skin temperature five to ten centimetres in diameter. Yet, according to Travell and Simons (1983:17), Kohlraush and Ruhmann reported diminished skin temperature in the vicinity of muscular nodules.

2.11 DIAGNOSIS

The diagnosis of IBFS will be confirmed by the following tests :

2.11.1 Noble's Compression Test :

The athlete is supine with knees flexed to ninety degrees. Pressure is placed over the proximal part of the lateral femoral epicondyle. The knee is gradually actively extended. At thirty to forty degrees of flexion, as the band moves over the lateral femoral epicondyle directly under the examiner's finger, the pain that the athlete feels during activity is reproduced (Noble, 1979; Noble, 1980 and Noakes, 1992: 450). The pain is located over the lateral femoral epicondyle and the lower third of the iliotibial band (Holmes et al. 1993). Noakes and Granger (1990: 97) state that the test may be negative if the runner has not run for a couple of days. This is due to the fact that signs and symptoms of IBFS subside with rest and therefore clinical examination is more relevant if performed shortly after activity (Martens et al. 1989).

2.11.2 Ober's Test

A positive Ober's test indicating a tight iliotibial band (Lindenberg et al. 1984) is associated with tensor fasciae latae trigger points (Travell and Simons, 1983: 222). For this test the patient is in the side-lying position, with the affected side uppermost. The patient flexes the lower leg at the hip and the knee to stabilize the body. The examiner then passively abducts and extends the patient's upper leg with the knee straight or flexed to ninety degrees. The examiner slowly lowers the lower limb and if a constricture is present it will remain abducted and will not fall to the bed. The hip is extended slightly so that the iliotibial band passes over the greater trochanter of the femur. Ober originally described performing the test with the knee flexed, which is easier for the examiner and in the neurologically involved patient population was adequate for a positive test. However, in the young active patient the test may be performed with the knee extended, which places more stretch on the iliotibial band and is more likely to reveal subtle tightness (Noble et al. 1982; Lindenberg et al. 1984 and Magee, 1992:354-355).

2.11.3 Renne (1975) described a manoevre in which the athlete stands on the affected leg with the knee flexed thirty to forty degrees. This position brings the iliotibial band in tight contact with the prominence of the lateral femoral epicondyle and may induce pain. This may be further accentuated by having the patient hop in knee flexed position.

According to Sutker et al. (1981); Sutker et al. (1985) and Martens et al. (1989) diagnosis is entirely clinical since radiographic examination is generally unremarkable.

2.11.4 Sandman (1981) regards pain as the most important criteria in trigger point diagnosis. The reason for this is that standard diagnostic procedures, laboratory work and radiographic studies show no signs of bone or joint pathology and there are no measurable metabolic changes (confirmed by Yunus et al. 1988). Thus the diagnostic guidelines to follow are the patients pain, distribution of pain, palpatory examination finding, and limitation of movement or restriction of motion of the affected areas.

A thorough history is therefore a vital aspect in the diagnosis of trigger points, and must reveal the nature, extent and location of the pain (Fischer, 1987). The diagnosis is difficult due to the fact that the majority of factors involved tend to be subjective. Bearing this in mind, a list of characteristics, all of which must be present in order to diagnose a trigger point, has been devised by Travell and Simons (1983: 18-19):-

- either a history of rapid onset during or shortly following acute overload stress, or a history of gradual onset with chronic overload of the affected muscle.

- a pattern of pain referred from the trigger point that is characteristic for that muscle in which it is located.
- weakness of affected muscle with associated restriction in its stretch range of motion.
- a taught, palpable band in the affected muscle.
- intense local tenderness of the taught band to applied pressure.
- a local twitch response provided by dry needling or snapping palpation of the trigger point.
- reproduction of the characteristic pain patterns (Bonica, 1957 and Simons, 1976).
- elimination of the clinical presentation by specific trigger point therapy i.e. to the tensor fasciae latae or iliotibial band.

A site of local tenderness is essential to the diagnosis, but non-specific. A local twitch response and pain reproduction when present, are specific and strongly diagnostic of the myofascial trigger point (Travell and Simons, 1983-19).

In order to achieve an objective assessment of the diagnosis of trigger points, Fischer (1987) advocates pressure threshold measurement using an algometer for a quantitative measurement of local tenderness. Fischer (1987) states that this method has been useful for diagnosis of tender spots and trigger points and their clinical management, particularly in the assessment of treatment results. Pressure threshold measurement is useful in diagnosis, follow up and evaluation of treatment results of muscular, ligamentous, tendinous and periosteal trigger points.

Other objective methods which may be used are a goniometer to assess for loss of ranges of motion (Sandman, 1981); EMG studies to show absence of electromyographic activity within the tense muscle fibres (Simons, 1976) and

thermography to document vasomotor disturbance in the area of the trigger point (Fischer, 1987). According to Simons (1988) thermographic changes can often, but not always substantiate active myofascial trigger points that have been identified by history and physical findings.

According to Travell and Simons (1983:222-223), trigger points in the superficial tensor fasciae latae muscle are disclosed with flat palpation of the muscle with the patient in a supine position. The muscle can be located by palpating the area while the patient internally rotates the thigh against resistance. When the patient is fully relaxed, the muscle is placed under slight stretch or tension. Palpation at right angles to the direction of muscular fibres reveals tight bands and a spot of maximum tenderness (i.e. trigger points) in each band. Pressure on active tensor fasciae latae trigger point sustained for up to ten seconds augments the pain referred from the trigger point. Snapping palpation of an active trigger point in a muscle, usually elicits a "local twitch" response.

Trigger points in the tensor fasciae latae may occur as a single muscle syndrome, or commonly develop secondary to trigger points in the gluteus minimus and sometimes the rectus femoris, iliopsoas and sartorius muscle groups (Travell and Simons, 1983:223).

2.12 DIFFERENTIAL DIAGNOSIS OF ILIOTIBIAL BAND FRICTION SYNDROME

According to Renne (1975); Jones and James (1987:280); Noakes (1992:449); and Holmes et al. (1993), the pain associated with IBFS is a severe pain on the lateral knee, directly over the lateral femoral epicondyle. Therefore, is the pain is a more diffuse pain near the joint line, consider the following conditions (Noble, 1985 and Holmes et al. 1983):

2.12.1 Popliteal Tendonitis

This overload injury causes lateral knee pain, especially on downhill running. Localized tenderness presents over the popliteus tendon. Pain is increased with knee flexion to ninety degrees abduction and external rotation of the hip (Noble, 1985:30-31).

According to Noble (1980), Mayfield describes popliteus tendon tenosynovitis as pain localized to the lateral aspect of the knee on weight bearing during fifteen to thirty degrees of knee flexion and aggravated by downhill running. Yet, Noble (1980) states that tenderness just anterior to the fibular collateral ligament (i.e. over the tendinous portion of popliteus) separates a similar clinical presentation from that of IBFS. Also radiologically radio-densities in the area of popliteus tendon may be found.

2.12.2 Lateral Meniscal Tear

This is due to rotational overloading of the lateral meniscus. It is especially seen in rugby, soccer, hockey, tennis and squash players. Pain is usually localized over the lateral knee but may radiate upwards and downwards. Pain is increased by full flexion and extension as well as locking of the knee (Noble, 1985:59-63).

2.12.3 Biceps Fermoris Tendonitis

This condition is especially seen in endurance sports for example, running and cycling. It is especially seen in runners due to the jarring element. The patient presents with pain in the region of the head of the fibula (Noble 1980 and Noble, 1985:34).

2.12.4 According to Renne (1975) and Noble (1980), Simillie describes a snapping iliotibial band from abnormal attachment to the patella. Renne (1975) and Noble (1980) also quote Hendryson who describe bursitis in relation to the fibular

collateral ligament. According to Noble (1980) both conditions have localized tenderness at the joint line level but lower than the IBFS.

Also, the possibility of the following conditions must also be considered: Patellar Tracking Dysfunction, lateral facet irritation, discoid meniscal cysts, lateral meniscal or capsular stains or avulsions, Lateral Patellar Compression Syndrome, patellar subluxation or dislocation, Patello-femoral Pain Syndrome, Patello-femoral Pain Syndrome, popliteus muscle inflammation, lateral ligamentous stains or avulsions, Degenerative Joint Disease, related metabolic conditions (e.g. Gout or Pseudogout) fracture or pathological conditions of the iliotibial tract (Orava 1978; Sutker et al. 1981; Sutker et al.1985; Jones and James, 1987:281; Reid, 1992:427 and Holmes et al. 1993:427).

2.13 DIFFERENTIAL DIAGNOSIS OF TENSOR FASCIAE LATAE TRIGGER POINTS

Travell and Simons (1983:221) state that referred pain patterns from tensor fasciae latae trigger points can easily be mistaken for the following conditions:

- L4 peripheral neuropathy
- peripheral nerve entrapment of meralgia parasthetica

Tensor fasciae latae trigger points also may be mistaken for trochanteric bursitis. But in disease of the bursa there is no referred pain pattern (Travell and Simons, 1983:221).

According to Travell and Simons (1983:221) tensor fasciae latae trigger points may produce a pain pattern confusingly similar to trigger points in the following muscles:

- gluteus minimus
- gluteus medius
- vastus lateralis

- quadratus lumborum muscle

2.14 TREATMENT

2.14.1 Myofascial Trigger Point Therapy

2.14.1.1 Dry Needling

Dry needling produces no anaphylactic response (Lewit, 1979 and Garvey et al. 1989). Conclusions drawn by Baldry (1989:36), after observation of various forms of invasive treatments, state that it makes no difference as to what substance, or for that matter, whether any substance, is inserted into trigger points. The reason for this is that the effectiveness of such procedures depends entirely on one factor common to all types of injections, namely, the mechanical irritation of the needle itself. These views are supported by Lewit (1979); Sola (1981) and Garvey et al. (1989).

According to Melzack (1981) treatment of myofascial trigger points is directed toward interrupting or preventing abnormal neural circuits responsible for the self-perpetuation of the pain-spasm pain-cycle. The needle mechanically disrupts the dysfunctioning nerve endings or contractile elements of the muscle which are thought to sustain the trigger point activity. The effect of needling thus results in relief of the patient's symptoms (Travell and Simons, 1983:34 and Garvey et al., 1989).

According to a special review by Simons (1976), there are thirty five recommended treatments for MFPS which are divided into six categories: physical therapy, injection, spray and stretch medical management, drugs and surgery. Of these, the treatments that were repeatedly reported to provide marked pain relief were massage, procaine injection and ethyl chloride spray. Mance et al. (1986) rate these same three treatments as the most common and effective, with injection

therapy being the most widely used of the three.

The technique of invasive needling, as described by Sola (1981) and Travell and Simons (1983:84-85), is consistent for dry needling and for injection of substances. The trigger point is located by palpation. The needle is inserted one to two centimetres away and directed towards the trigger point such that the needle approaches the trigger point at an angle of about thirty degrees to the skin. When the needle penetrates the trigger point, the patient usually experiences the referred pain and twitch response of the involved muscle. A fanning technique is used, where the needle is repeatedly withdrawn out of the trigger point and re-directed to penetrate a new part of the trigger point. The site is then re-palpated for any remaining tender spots. If one is found, it is accurately localized with the fingers and is needled using the method described above. All tender spots in the region should be eliminated before the needling procedure is considered complete.

Baldry (1989:51) states that when a needle is inserted into trigger point, superficial vessels around it often become dilated, presumably as a result of histamine release, and as a result of this, a raised papule or a more diffuse area of redness may develop together with a sensational warmth.

According to Travell and Simons (1983:225) myofascial trigger points in the tensor fasciae latae muscle are needled with the patient lying supine. The muscle is identified by asking the patient to internally rotate the thigh. To localise the taught bands it may be necessary to slacken the muscle slightly by placing a pillow under the knee thus flexing the hip. When trigger point tenderness has been precisely located, pressure is applied with fingers of one hand to pin down the taught band as the needle inserted into its trigger points with the other hand. A local twitch or "jump sing" is often observed (Travell and Simons, 1983:225).

If the tensor fasciae latae muscle has been accurately identified, no major nerves or vessels lie in the path of the needle, which is angled nearly horizontally to penetrate this subcutaneous muscle. After needling, the patient should then actively move the thigh slowly through the full flexion - extension range of hip motion.

Levine et al. (1976); Lewit (1979); MacDonald et al. (1983); Mendelson et al. (1983); Baldry (1989:36) and Garvey et al. (1989), found dry needling to be more effective than procaine injection. These views are rejected by Good (1951), Simons (1976); Yunus et al. (1981); Simons (1983) and Murphy (1989). According to Murphy (1989), the best results are achieved by using a combination of electrotherapies (ultrasound high-voltage stimulation or transcutaneous electrical nerve stimulation) and spray and stretch techniques. Murphy (1989) also states that, in opinion of the clinicians, dry needling produces the same results as an anaesthetic injection, but he personally had more consistent success with the latter.

The results of study performed by Garvey et al. (1989) suggested that the injected substance is not the critical factor in trigger point therapy. The dry needle group showed a better rate of improvement than two groups receiving anaesthetic injection, re-enforcing the belief that the mechanical effect of the needle is the important factor in the deactivation of trigger points (Garvey et al. 1989).

These results are supported by Lewit (1979), who states that dry needling is highly effective in the treatment of chronic myofascial pain. Of the three hundred and twelve patients treated by Lewit, immediate analgesia was produced by dry needling in 86.6% of cases. The needle effect (immediate analgesia without hyperaesthesia) may be produced by precisely needling the most sensitive spot of the trigger point. He further states that the long-term therapeutic effects

previously ascribed to local anaesthetics may in fact be due to needling. Further support for dry needle therapy comes from MacDonald et al. (1983) and Mendelson et al. (1983) who found that superficial acupuncture was effective in the treatment of low back pain.

Baldry (1989:37) states that needling with a solid, pointed needle should be the treatment of choice over any treatment using a hypodermic needle (saline or anaesthetic injections). The reason for this is that the cutting edge of the hypodermic needles causes microtrauma and damage to blood vessels, an undesirable effect in the treatment of MFPS. The pointed needles are thinner (they do not need to be hollow) and the likelihood of damage to blood vessels is far less.

Travell and Simons (1983:79-80) postulate several mechanisms that may contribute to the deactivation of trigger points by needling. The needle may mechanically disrupt the muscle fibres or nerve endings (which are sensory and motor components) and, therefore, the feedback cycle that sustains abnormal muscle activity. The needle may also disrupt the muscle fibres and result in the release of intracellular potassium which could cause a depolarization block of the reverberating nerve fibres. Injected fluid helps to "wash out" or dilute any nerve-sensitizing substances and thus reduce the irritability of the trigger point. According to Sola (1984), the medical history should screen for patients who report hypersensitivity and syncopal reactions to injections. Sola (1984), states that patients with fair skin, light or red hair and blue eyes have a greater tendency towards these reactions and may experience more flare ups and pain after treatment. He states that these patients should not have more than a few points needled at the first visit. It is appropriate to watch for signs of fainting and this can be monitored by touching the patient's skin with the back of one's hand to detect for excessive sweating. Bleeding into tissues may occur after treatment. If this

should occur. treatment should be followed by application of cold packs (Sola, 1984).

The main objections to injecting procaine into trigger points are due to its potentially undesirable side effects, which may be divided into those occurring at the site of injection and general effects. The intra-muscular injection of a local anaesthetic may cause muscle necrosis. Also, procaine, when injected into a muscle is capable of providing a distressing, yet temporary loss of power. Further, when injected around a nerve it is liable to cause some transitory sensory loss (Baldry, 1989:36). Systematically, procaine injections may cause allergic reactions and close-related toxic reaction. The most serious allergic reaction to this drug, anaphylactic shock, although extremely rare is nevertheless life threatening when it occurs.

Sola and Williams (1956) and Travell and Simons (1983:80) state that even mild hypersensitivity to local analgesics may cause light-headedness, palpitations and mild hypotensive reactions.

Levine et al. (1976) states that the needling technique's effectiveness was strongly influenced by the placebo effect. It was found that high anxiety and depression tests scores as well as good doctor-patient rapport were significant predictors of analgesia derived from needling.

Sandman (1981) states that nutritional supplementation and daily stretching exercises hasten recovery. According to Bennett (1986), treatment with non-steroidal anti-inflammatory drugs and even corticosteroids are ineffective therapies for myofascial trigger points.

Travell and Simons (1983:18) state that several treatments may be required for

elimination of active trigger points. Incomplete restoration of the normal full length of the muscle usually means incomplete relief of pain referred from its trigger points.

If, within a few minutes to a few hours after treatment, the patient experiences severe cramping pain in the general region of treatment, shortening activation of an antagonist muscle may have occurred (Travell and Simons, 1983:73). Martin (1952); Lewit (1979) and Sola (1991) state that after immediate relief of needling, a reactivation of pain may occur several hours later on the following day. He stated that this usually lasts for one or two days, and only then is full therapeutic effect observed. According to Travell and Simons (1983:65), increased local soreness for several days after trigger point injection is due to a vitamin B or ascorbic acid insufficiency. this may exacerbate rather than relieve the pain referred from active trigger points remaining in the muscles injected.

2.14.1.2 Stretch of the Tensor Fasciae Latae Muscle

Firer (1989) and Baker (1995) recommend stretching in the treatment and prophylaxis of IBFS. Jones and James (1987:283) state that this is extremely important, especially in those individuals with a positive Ober's test.

Bonica (1957); Sandman (1981) and Travell and Simons (1983:85) emphasize that stretch techniques are important in returning the muscles to their normal maximum length. Without stretching, trigger point therapy would not be nearly as effective. Passive stretching may even inactivate a trigger point without the application of any other form of treatment (Travell and Simons, 1983:65:89). Lewit and Simons (1984) promote post - isometric relaxation (manually resisted isometric contraction followed by relaxation) as a simple, harmless, non-invasive\ve and effective way of restoring full stretch length to muscles containing trigger points. Stretching may be facilitated by the application of cold, due to the

fact that cold can decrease muscle spasm (Prentice, 1986:82-83). According to Noakes and Granger (1990:100), stretches should be performed for ten minutes daily. Travell and Simons (1983:18) state that relief of pain as a result of myofascial therapy is more likely to be lasting when the patient moves the treated muscles through several cycles of their full range of motion at the end of therapy sessions.

Sandman (1981) and Travell and Simons (1983:66-67) state that the effected muscle should be stretched slowly, with steady gradually increasing force. The stretch is then held at a point of tolerable discomfort. Jerking the muscle or sudden movements by the patient must be avoided during and after the stretch. Travell and Simons (1983:67) state that reaching the full normal length of the muscle is essential for complete inactivation of its trigger points and relief of referred pain. Achieving the final few degrees of movement is critical. Sandman (1981) states that a programme of daily stretching exercise at home should be added to the patient's therapy programme.

According to Noble (1982) and Travell and Simons (1983:223-224), the following position is sued to stretch the tensor fasciae latae muscle.

The patient lies on the unaffected side. The uppermost thigh is abducted. To prevent the tensor fasciae latae from painfully impinging on the greater trochanter as it is lengthened, the clinician first extends the abducted thigh and then adducts it. The clinician's hand should be placed so that it stabilizes the lumbar spine and pelvis by holding the knee of the untreated limb on the examining table i.e. the unaffected leg is flexed to reduce the lumbar lordosis. According to Reid (1992:425), a variation of the same exercise is as follow:

The patient lies on their side with their back near the edge of the examination table

with the affected side facing upwards. The unaffected leg is flexed. The painful leg is held in extension at the hip and the knee is allowed to hang over the edge of the table and therefore passively adducts under the influence of gravity. This position is held for fifteen minutes with the rest periodically to prevent discomfort. This is a passive form of stretch and can be used at the initial onset of IBFS>

According to Jones and James (1987:283) and Reid (1992:426), the iliotibial band can be stretched while the athlete is standing. The patient stands with both knees in full extension and extends and adducts the affected leg far as possible. The trunk is then flexed laterally as far as possible towards the unaffected side. Alternatively the patient may rotate the waist away from the affected side flexing the trunk and attempting to touch the heel of the affected leg with both hands. The stretched position is held for a count of thirty seconds. The exercise is repeated five times (See Appendix 12A).

Another exercise can be performed while lying on the side, the normal leg uppermost and the hips extended in a straight line with the trunk. Patient flexes at the waist, pushing themselves up into a resting position on hip of the affected side, placing their hand directly under the shoulders and bearing weight on the extended arm and hand. It is essential to maintain back extension and to maximize tension on the iliotibial band. It is sometimes necessary to place the opposite foot on the floor to stabilize the pelvis. The position is held for ten seconds and is repeated five to ten times. (Noble, 1982 and Reid, 1992:426-427). Another stretch for the Iliotibial Band can be done while sitting. Both hands are placed on the injured knee, which is pulled across the body to the opposite armpit (Noakes and Granger, 1990:100).

Travell and Simons (1983:18) and Murphy (1989) state that exercises of the affected muscle, along with patient co-operation greatly enhances the long term

effectiveness of trigger points therapy. The adequacy of corrective action (i.e. what the patient must do or avoid, including stretching, to achieve lasting recovery) usually determines the duration of relief experienced after treatment of the involved muscles (Graff Radford et al. 1987).

2.14.1.3 Cryotherapy - The Effect of Ice on Iliotibial Band Friction Syndrome

Orava (1978); Noble (1979); Noble (1980); Noble et al. (1982); Firer (1989) and Baker (1995), recommend ice in the treatment of IBFS. Ice is also effective in the reduction of pain associated with myofascial trigger points (MacDonald, 1980; Rubin 1981 and Sola, 1981). Injury to muscular or tendinous tissue, as in IBFS is associated with a local inflammatory reaction (Noble, 1980; Lindenberg et al. 1984; Martens et al. 1989 and Noble, 1980). According to McMaster (1982) and Prentice (1986:82) ice is used to reduce many adverse reactions related to inflammation i.e. ice decreased oedema, inflammation and local anaesthetizing effect on pain. McMaster (1982), Prentice (1986:82) and Sola (1991) state that a moist pack applied to the area of pain relieves the temporary discomfort of needling. The extent to which the tissue is cooled depends on the cold medium that is being applied, the length of cold exposure and the conductivity of the area being cooled. In most cases, the longer the cold exposure is, the deeper the cooling. At a temperature of three and a half degrees celsius, muscle temperatures can be reduced as deep as four centimetres (Arnheim and Prentice, 1993:310).

The physiological effects of ice are as follows:

According to Arnheim and Prentice (1993:310), when cold is applied to the skin for fifteen minutes or less at a temperature of ten degrees celsius or less, vasoconstriction of the arterioles and venules in the area occurs. This vasoconstriction is caused, in part, from simulation of the sympathetic nervous

system and adrenal medulla, causing secretion of norepinephrine and epinephrine.

Much of the damage observed in cells following trauma occurs as a result of compromised circulation, which decreases the amount of oxygen delivery to cells in the area of injury. The immediate use of ice following injury decreases the extent of hypoxic injury to those cells on the periphery of the primary injury by slowing their metabolic rate. This results in less damage to the tissues, thus decreasing rehabilitation time (Knight, 1995:9-11). Because cold lowers the metabolic rate and produces vasoconstriction, swelling will be reduced (Arnheim and Prentice, 1993:310).

According to Prentice (1986:82), cooling tissues can directly decrease muscle spasm in the area, thus decreasing waste products which act as a muscle irritants. Such waste products or accumulation of metabolites facilitate trigger point formation (Awad, 1973; Sandman, 1981 and Mance et al. 1986). Thus, ice aids in the alleviation of pain due to the presence of myofascial trigger points (MacDonald, 1980; Rubin, 1981 and Sola, 1981). A muscle spasm can also be decreased when cold increase muscular viscosity, slowing its ability to contract (Arnheim and Prentice, 1993:311). Because the local application of cold can decrease acute muscle spasm, the muscle becomes more amenable to stretch (Knight, 1995:233-239). Cold also decreases free nerve ending excitability of peripheral nerves (Prentice 1986:83).

Thus, in the treatment of IBFS, cryotherapy results in decreased inflammation of the iliotibial band as it frictions across the lateral femoral epicondyle. Icing results in decreased blood flow and decreased pain perception. Cryotherapy leads to decreased muscle spasm in the tensor fasciae latae muscle and iliotibial band due to decreased metabolic rate and also aids in the alleviation of myofascial trigger

points. Melzack et al. (1977) quote Travell and Rinzler who state that a brief intense stimulation of trigger points by intense cold, diminishes or abolishes pain for days, weeks and sometimes permanently.

McMaster (1982); Prentice (1986:89); Arnheim and Prentice (1993:311-312); and Knight (1995:180-193) state that although adverse reactions to cold are uncommon, they do happen and are described as follows :

i. Cooling for a hour at minus one degree Celsius produces redness and oedema that lasts for twenty hours after exposure. Frostbite has been known to occur in subfreezing temperatures of minus three to minus four degrees celsius.

ii. Exposure of ninety minutes at minus fourteen to minus sixteen degrees celsius can delay resolution of swelling for up to one week.

iii. Some individuals are allergic to cold and react with hives, joint pain and swelling.

iv. Paroxysmal cold haemoglobinuria is a rare disease that occurs minutes after cold exposure and may lead to renal dysfunction, secondary hypertension and coma. Early symptoms are severe pain in the back and legs, headaches, vomiting, diarrhoea and dark brown urine.

2.14.1.4 Patient Education

According to Noakes (1992:450-451), patient information should include the following information in order to decrease the perpetuating factors of IBFS:

2.14.1.4.1 Training

The patient will be advised to decrease training distances and to run only to the point of pain and never to the point were the injury becomes frankly painful (Noble, 1979; Noble, 1980; Sutker et al.1981; Noble, 1985:34 and Noakes and

Granger, 1990:100). It is recommended to run on soft surfaces (i.e. grass), to avoid downhill running and to switch sides of the road from time to time (Noble, 1980; Noble, 1985:34; Noakes & Granger, 1990:100; Noakes 1992:450-451 and Baker, 1995). Lindenberg et al. (1984) states that hill running especially should be avoided until the athlete has been asymptomatic for at least four weeks. Also, the patient should be instructed on the importance of warming up before exercise. Noble (1979); (1980), states that speed running should be avoided and shortening of running stride may also be helpful, although this has not been proven. He further also advises running on flat surfaces with some cushioning e.g. grass.

Holmes et al. (1993) recommended certain training modifications with respect to cyclists who present with acute IBFS. During the initial two weeks, rides should be done on flat terrains and mileage should be reduced by half. Further, cyclists should pedal easily at eighty revolutions per minute or less with little resistance for the first week during which time they should ride at a pain-free cadence. Any cyclist who experienced discomfort while using these training modifications were instructed to abstain from cycling for at least ten days. He stated that cyclists with chronic IBFS symptoms had more restrictive training modifications. Cyclists were to completely abstain from cycling or other sports - related activities for the initial two weeks. If symptoms persisted the abstinence period was extended to four additional weeks.

If symptoms then improved, the cyclist could return to low-mileage, low-resistance cycling on a flat terrain. Training practices could then resume only when indicated by a sports medicine clinician.

2.14.1.4.2 Shoes

The patient is advised to purchase soft running shoes with less motion control. Such shoes have soft midsoles, weak heel counters and very little resistance to vertical bending around the long axis. The concept is to encourage a slightly more

pronated position of the foot. (Lindenberg et al. 1984; Granger 1990:100; Noakes 1992:450; Reid 1992:425 and Baker, 1995). The aim is to increase the ability of the limb to absorb shock.

2.14.1.4.3 Bicycle Adjustments

For cyclists riding with a fixed pedal system, cleat or pedal position adjustments are recommended to reflect the normal off-bike alignment. For example, external tibial rotation of greater than twenty degrees required an externally rotated fixed cleat. In previous studies, saddles were adjusted for thirty to thirty two degrees of flexion of the knee at the bottom centre of the pedalling stroke (Holmes et al., 1993).

2.14.1.4.1 Stretches

Specific stretching exercises are recommended(See Appendix 12 A). Such exercises assist in stretching the iliotibial band and tensor fasciae latae and should be carried out as often as possible (Lindenberg et al. 1984). The aim is to increase the flexibility of the iliotibial band and thus decrease the incidence of iliotibial band friction over the lateral femoral epicondyle and thus decrease the signs and symptoms of IBFS.

2.14.2 Other Forms of Treatment

According to Firer (1989), acute injuries respond better than chronic injuries to conservative management. Other forms of treatment of IBFS include prescription-made orthotic inserts; hydrocortisone injections and surgery (Noble, 1979; Noble, 1980; Firer, 1989; Martens et al. 1989; Noakes, 1992:451, Reid, 1992:427 and Baker, 1995). Patients are also instructed to rest (Noble 1979; Noble 1980; McBryde et al. 1985:406; Jones and James, 1987:282, Firer 1989 and Baker 1995). Noble (1980) advises a four to six week period of rest if local treatment of icing and steroid injection fails. According to Noble (1980) and

Jones and James (1987:280), as IBFS is an overuse syndrome resulting in inflammation, most cases should subside on immediate cessation of activity. Yet, most runners set goals for themselves and nothing, even injury, can stop them from running. There is a tendency for some runners to attempt to run with pain and hope that it will subside. Thus, most runners will be prepared to accept a reduction in activity, which means running on a flat surface (without cambers or hills), reduction of distance and speed and cessation of running if pain occurs (Noakes, 1992:448-452). Other cardiovascular stimulatory activity such as swimming, may be brought in to supplement training (Noble, 1980). Another form of treatment is cross-friction applied to the tender area (Noakes and Granger, 1990:100). Oral anti-inflammatories may also be helpful in reduction of inflammation (Renne 1975; Noble 1980; Jones and James, 1987:282 and Baker 1995). Jones and James (1987:282) state that the physician must inform the patient that even the acute, milder cases may take as long as six weeks to resolve.

Jones and James (1987:283) urge athletes who have rested for a period more than three to four weeks to return gradually to their training programmes. Jones and James (1987:283) quote James, Bates and Osterning who state that the runner should begin jogging for fifteen minutes a day at a mild pace, and each subsequent week five minutes are added to the daily time until a cumulative time of forty minutes, non-stop without discomfort is reached. This will require six weeks, and if satisfactory progress has been realized, return to previous training regime is allowed.

2.14.2.1 Custom-Made Orthotic Inserts

It may be necessary for an orthopedic technician to build a lateral wedge into the midsole of the shoe for severe genu varum or very high arched rigid feet (Noakes, 1992:451; Noakes and Granger, 1990:100; Noble, 1982; McBryde et al. and Sutker et al. 1981) as genu varum leads to iliotibial band tightness (McBryde et al. 1989; Jones and James, 1987:282 and Reid 1992:425). Also, rigid cavus feet are unable to absorb shock adequately (Sutker et al. 1981; Noakes and Granger

1990:99 and Noakes 1992:450). Noakes (1992:451) quotes Kvist who states that a wedge is utilized to force the foot to pronate thus improving its shock-absorbing capacity by twenty five per cent. Noakes (1992:451) states that if an athlete has a short leg, the sole of the shoe on the short leg side should be built up to compensate for the shortness. Lindenberg et al. (1984) states that both inadequate and excessive pronation are considered aetiological factors of IBFS. Thus, orthotics should be utilized to resist or increase pronation depending on the foot structure of the individual runner. The fact that a small percentage of patients with IBFS were ultimately helped when they switched to hard running shoes with prescribed corrective orthotics to limit ankle pronation, the opposite of what was suggested initially, shows that the mechanics causing this injury are not fully understood (Noakes and Granger, 1990:101).

2.14.2.2 Hydrocortisone Injection

Hydrocortisone has detrimental side-effects and the benefits versus risks must be seriously considered before administering these drugs (Arky, 1996: 1670-1672). IBFS responds well to more conservative treatment (Orava, 1978 and Henderson, 1989) and therefore hydrocortisone injections are tried in more resistant cases (Noble, 1979; Noble, 1980; Firer, 1989; Noakes and Granger, 1990: 100; Noakes, 1992:451 and Reid, 1992: 427) The function of hydrocortisone is that it provides relief for inflamed areas in the body. In the case of IBFS, it lessens pain, swelling and redness. Side-effects include that it lowers the patient's resistance to infection and infection may be harder to treat. Other less common side effects include: decreased or blurred vision, frequent urination, increased thirst, rectal bleeding and burning or itching. Additional side effects may occur if this medication is taken over a long period of time. Side-effects not requiring medical treatment may occur including: increased appetite, indigestion, nervousness and insomnia (United States Pharmacopeia, 1996: 608-613).

Dosage of Hydrocortisone Sodium Phosphate varies between 5-75 milligrams. This is injected every two to three weeks at the site of pain (United States Pharmacopeia, 1996: 608-613).

2.14.2.3 Surgery

Surgery is based on the fact that at thirty degrees of flexion the posterior fibres of the iliotibial band abut the lateral femoral epicondyle. Also, on palpation the posterior fibres appear tighter against the lateral femoral epicondyle than the more anterior fibres (Noble, 1979; Noble, 1980 and Noble 1985:134). Firer (1989) and Martens et al. (1989) state that an alternative is needed for patients with complaints that are resistant to conservative means. Firer (1989) and Martens et al. (1989) state that not all cases can be cured by conservative means and that surgery is indicated if the patient is not willing to give up his sports activity. Resection of a small triangular piece (one to two centimeters) of the distal posterior band produced uniform good results, low morbidity and a quick return to sports (Noble, 1985:34 and Martens et al. 1989). Noble (1985:34) stated that runners can usually recommence running in four to six weeks. Yet, Orava (1978) and Henderson (1989) state that this syndrome responds well to conservative treatment in most cases and surgery is therefore rarely required. Noble (1979) and (1980) recommends surgery, but only after a local treatment of icing and steroid injection, as well as, a four to six week period of rest have failed. Initially, Noble (1980) performed a double procedure i.e. excision of the lateral epicondylar prominence and release of the posterior two centimetres of overlying iliotibial band. Later, surgery was limited to a soft tissue release by a transverse incision across the line of the fibres of the iliotibial band. Martens et al. (1989) observed no functional or clinical abnormality after the limited resection of the posterior part of the iliotibial tract of the knee in athletes.

CHAPTER THREE

MATERIALS AND METHODS

Materials and Methods

3.1 STUDY DESIGN AND PROTOCOL

This controlled placebo study proposed to investigate the effects of MFTPT of the tensor fasciae latae muscle and iliotibial band in patients presenting with IBFS. The objectives were to assess each of the two treatment groups (i.e.. patients who received MFTPT and those who received "sham" or placebo treatment) for intra-group improvement. Once this had been achieved an intergroup statistical analysis determined which of the two treatments, if any, was more effective. The aim was to establish the efficacy of MFTPT on IBFS

The sample size was set at thirty patients which were divided into two groups of fifteen by random assignment. The method of random assignment of patients to experimental groups was done as follows. Six groups of five were formed. These were numbered from one to six which represented a particular order of control or experimental, i.e.- 1) CEECC; 2) CECEE; 3)CCEEE; 4)ECCEC; 5) ECECC; 6) EECCE. "C" was representative of the control group and "E" was representative of the experimental group. A dice was thrown five times to determine the order of experimental versus placebo patient. The two groups of fifteen were the "control group" and the "experimental group".

The methodology used was similar in execution to that used by Jones (1994). The procedure for the study was as follows. Each patient that passed the initial screening was scheduled for a two hour consultation where a case history (Appendix 1); physical examination (Appendix 2), knee, as well as hip regional examination (Appendix 3 and 4) were completed. Once this was completed and the patient's condition had satisfied the diagnostic criteria, the patient was treated for a maximum of ten treatments over a period of one month. Lindenberg et al. (1994) advocates 83% (thirty of thirty-six) of patients with IBFS treated conservatively were completely cured and 58% (twenty-one of thirty-six) were completely cured within three weeks.

The diagnosis of IBFS was explained to the patient. Consultations served to administer treatment, monitor progress and compliance, reinforce exercises and address and control any other perpetuating factors.

3.2 SUBJECTS

During the year 1995 extensive advertising was undertaken to acquire subjects for the study. Thirty patients fulfilled the requirements and were selected, ie : they presented with severe pain over the lateral knee and tenderness of the lateral epicondyle of the femur for

two to four centimetres that was associated with running. They also presented with tenderness on palpation in the tensor fasciae latae and iliotibial band.

Patients taking anti-inflammatories (excluding topically applied anti-inflammatories) for their condition or suffering from any systemic disease were excluded from the study. The patient group consisted of those being diagnosed with IBFS with active myofascial trigger points, associated with Myofascial Pain and Dysfunction Syndrome (MPDS) in the tensor fasciae latae muscle and/or the iliotibial band. (For diagnosis of symptoms and signs of IBFS as well as criteria to diagnose MPDS refer to Literature Review).

3.3 ETHICS

The procedures that followed were in accordance with the ethical standards of the Responsible Committee on Human Experimentation. Patients were informed beforehand that they would be taking part in the study and thus had the chance of withdrawing before commencing the research programme. Each patient also signed a letter of consent prior to commencement.

3.4 INTERVENTION

During the first two weeks, three treatments were administered per week and during the last two weeks, two treatments per week were administered. This is in accordance with Mance et al. (1986) who specified that trigger point injection should be administered every second, third or fourth day. A follow up consultation took place one month later. If the patient could run at pre-injury symptom-free level before the completion of ten treatments, they were asked to have two more treatment sessions. If the patient was still symptom free at this stage they no longer received treatments. The re-evaluation criteria however, still took place one month later.

Once located, the trigger points were drawn onto the pain drawing (Appendix 7) as a representation of the patient. This ensured that at subsequent consultations, the exact locations of specific trigger points, could be localized, which would facilitate accurate relocation.

The experimental group received authentic treatment in the form of dry needling, cryotherapy and stretching. The patients were expected to keep up their programme of daily stretches during the entire treatment and follow-up period. A number 8 (30mm long, 30-gauge) stainless steel acupuncture needle was utilized, the same used by MacDonald et al. (1983). Once the trigger points had been accurately located in the iliotibial band and tensor fasciae latae muscle and the

overlying skin was cleaned with alcohol, the needle was inserted into the trigger point. To minimize the pain of insertion each needle was inserted swiftly. This is in accordance with MacDonald et al. (1983).

Chemical cold packs were utilized for members of the experimental group due to their ease of application. These packs did not make a mess, were cheap and could be refrozen after use. These cold packs were placed over the site of inflammation where the iliotibial band crosses the lateral femoral epicondyle as well as trigger points in the tensor fasciae latae and iliotibial band.

At this point the pain was often recreated in its referral pattern by the action of the needle. The needle manoeuvred in such a manner as to pierce the trigger point and then withdraw to a point such that the tip of the needle remained inserted in the skin but the trigger point was free of the needle. The angle of penetration was changed and the needle re-inserted into the trigger point. This procedure was repeated so that a fan-shape of repeated penetration was achieved, assuring maximum coverage of the area of the trigger point. This technique was consistent with that advocated by Bonica (1957); Sola (1981); Travell and Simons (1983:84-85); Sola (1984) and Sola (1991) and it served to deactivate the trigger point. A new needle was used for each treatment and for each trigger point. Each trigger point located was treated at each consultation (Patients had trigger point numbers varying between one and seven).

The placebo group received a sham treatment similar to the method used by Jones (1994). This was in the form of detuned ultrasound. (The ultrasound machine used was a Sonoplus 430 supplied by Mediotronics and made by Enraf Nonius). The "timer" of the ultrasound machine was set but the unit was turned off before treatment began. In this way the group believed they were getting treatment. The experimental group received patient education so as to decrease the causative factors of IBFS.

Stretch exercises (Appendix 12A) were taught to patients of the experimental group. Patients were instructed to hold each stretched position for ten seconds and to repeat each stretch five times during each of the three daily sessions. Patients were expected to keep up this programme of daily stretches during the entire treatment and follow up period.

Data was obtained in the following way :

Primary data was obtained by written communication. The Numerical Pain Rating Scale 101 (Appendix 5) McGill Pain Questionnaire (Appendix 6), Pain Drawing (Appendix 7) and Pain Disability Index (Appendix 8) were completed. These were utilized to record the response of the patient in a subjective manner.

Measurement of pain sensitivity of trigger points to pressure was provided by an algometer (Model fdk20 force dial made by Wagner Instruments and supplied by Activator Methods Inc) (See Appendix 10A, 10B and 10C) and used to record the response of patients in an objective manner.

Secondary data collected were journal articles, published reports and books containing information relevant to the research being conducted. It also included a case history, physical examination and regional knee and hip examination forms (Appendix 1-4) which are used in Technikon Natal Chiropractic Day Clinic.

The sample of patients included those residing in the greater Durban area which therefore eliminated the possibility of poor compliance due to travelling or transport problems.

The following were criteria governing admissibility of the data :

i) Only data from the relevant questionnaires that were completed under the supervision of the researcher was used.

ii) Only data was collected from patients who were eligible for the study by satisfying all the conditions. The following criteria were taken into consideration before accepting patients into the study.

- Patients suffering from any systemic disease were excluded from the study.
- The study excluded patients taking anti-inflammatories for their condition, yet, patients taking topically applied anti-inflammatories were not excluded.

iii) Only objective data collected from reputable mechanical devices was used. i.e. : the algometer. All algometer readings were determined solely by the principal investigator.

iv) All treatments were conducted by the principal investigator.

v) Only data obtained from references of high academic repute were used.

The detailed patient procedure is as follows :

a) The initial consultation

A full case history, a physical examination (including knee and hip regional examinations) (Appendix 1-4) were performed so as to give the precise diagnosis. When taking the case history, certain questions were emphasized. The following questions were adapted from Lindenberg et al. (1984)

- How many years have you been running? (i.e. running experience)
- What is your running speciality? (i.e. Non-competitive, competitive, marathon or short distance running?)
- What is the average distance you ran eight weeks before your injury?
- What type of training do you do? Long slow distance, interval training or fast sustained effort?
- On which type of surface do you train? (e.g. road, dirt or grass)
- Have you had recent changes in your training schedule? Have you increased your distance, speed or your hill running?
- What type of running shoes were you using before and after your injury?
- How much time did you spend stretching each day?
- Did you wear special shoes or arch supports as a child?
- How long have you had symptoms?
- Have you had any other treatment for your condition? Who treated you? Did it help?

Questions were also asked to determine the grade of the injury (see Appendix 13). This was adapted from Reid (1992: 424-429). It is also very important to note the patients age to determine which age category the injury is predominate in.

When undertaking the physical examination, the following in particular were observed: Short, leg, genu varum and valgum; high arched or flat feet and alignment of the foot.

To determine whether the patient had a short leg, a tape measure was utilized to measure the distance between the anterior superior iliac spine and medial malleolus bilaterally. The test was then repeated by measuring from the umbilicus to the left

medial malleolus and then from the umbilicus to the right medial malleolus. A difference of greater than one centimetre between the left and right side indicated a leg length discrepancy (Magee, 1992:423).

Genu varum was measured by simply measuring the distance between the knees with the patients standing with the knees extended and feet together. This is in accordance with a study performed by Noble (1980). Patients were defined as having genu valgum if there was a space between the medial malleoli when the medial femoral condyles were touching while standing (Magee, 1992:376).

The Feiss Line Test was used to determine the extent of pes planus development. The apex of the medial malleolus was marked as well as the plantar aspect of the first metatarso-phalangeal joint. The navicular tuberosity was then palpated and its position was noted with respect to the line joining the medial malleolus and the plantar aspect of the first metatarsophalangeal joint. The patient then stood with the feet eight to fifteen centimetres apart. The navicular tuberosity should have been positioned on the line joining the two points. If it dropped, this indicated flat feet (Magee, 1992:484).

Foot alignment indicating pronation and supination was noted merely by visual inspection.

Runners shoes were examined for wear patterns and resilience of the midsole was checked along the angle of the heel flare. This was done by the examiner compressing the heel flare. This was adapted from Lindenberg et al. (1984).

Once this was completed and the patient's condition satisfied the diagnostic criteria, the patient was allotted via the described method, to either the control or experimental group. The McGill Pain Questionnaire, Numerical Pain Rating Scale 101, Pain Drawing and Pain Disability Index were completed and algometer readings taken. This was followed by treatment of the patient.

b) Treatment Consultations

The McGill Pain Questionnaire, Numerical Pain Rating Scale 101, Pain Drawing and Pain Disability Index were completed and algometer readings taken prior to the sixth and tenth treatment. This was followed by treatment of the patient.

c) Follow up consultations

The follow-up consultation took place one month later following the final treatment. The McGill Pain Questionnaire, Numerical Pain Rating Scale 101, Pain Disability Index and Pain Drawings were completed and algometer readings ascertained. No treatment was given.

3.5 MEASUREMENT, OBSERVATIONS AND STATISTICAL ANALYSIS

Numerical Pain Rating Scale 101 was based on the example set by Jensen et al. (1986). It was chosen because of its ease in application when providing subjective information on the levels of pain as well as its established validity and reliability. Patients were asked to rate their perceived level of pain intensity on a numerical scale from 0-100, with the O representing one extreme (e.g.. "no pain") and the 100 representing the other extreme (e.g. "pain as bad as it could be"). The number stated by the patient as representing their pain intensity is the basic data for the Numerical Rating Scale 101 (Jensen et al. 1986). It was therefore used to monitor the progress of the patient, as a decrease of pain intensity as felt by the patient, is indicative of an improvement of their condition.

The Short Term McGill Pain Questionnaire was used in this study (Melzack and Katz, 1992:164). It was used to gather further information concerning the intensity of pain. Its use along with confirmation of its reliability, validity and consistency are provided by Melzack and Katz (1992:152-164). The answers were ranked on an intensity scale that can be analyzed statistically once all questionnaires had been completed. This questionnaire was divided into two sections. Questions 1 to 11 represented the sensory dimension of pain experienced and question 12 to 15 represented the affective dimension. Each question scored a maximum of 3 for the most severe symptoms in that particular category, and a minimum of 0 for no symptoms in that particular category. The sum of all completed sections was calculated and given as a percentage of the highest possible score. If all sections were completed the highest possible score was 45, and would decrease by 3 for each section not completed.

The Pain Disability Index provided subjective information as to the extent to which the patient's pain influenced their normal daily activities. The indices used in this study were constructed the Neck Disability Index (Vernon and Mior, 1991), The Oswestry Low Back Disability Index (Fairbank et al. 1980) and The Classification of Injury Grade for Overuse Syndromes (Lindenberg et al. 1984 and Reid, 1992:425)). This Pain Disability Index was devised by a combination of the above due to the fact that no pain index deals with the knee region. Before using the devised Pain Disability Index it was pre-tested on a sample of thirty people. Once the questionnaire was judged acceptable it was submitted for use.

The aim of this index was to determine the extent that IBFS interferes with daily living.

Answers to the questionnaire were scored and statistically analyzed to ascertain the effectiveness of the treatment for each section, the highest possible score is 5 and the lowest was 0. The top statement scored 0 if it was marked and the score progressively increased by 1 for each statement marked below up to a maximum 5 if the last statement was marked. The sum of all the completed sections was calculated and given as a percentage. If all the sections were completed, the highest possible score was 60 and would decrease by 5 for each section not completed.

The statistics used to evaluate and capture data of subproblem one (The Numerical Rating Scale, McGill Pain Questionnaire and Pain Disability Index) was Wilcoxon's paired signed rank test at the 5% level of significance, for intra-group analysis. The tests were done using the computer software programme STATGRAPHICS PLUS VERSION 6 supplied by MANUGISTICS INC. The processed data was then presented in tabular format for easy interpretation.

An algometer may be defined as an apparatus for determining sensitivity to pain caused by pressure. According for Fischer (1987), pressure threshold measurement is used for the evaluation of the therapeutic efficiency in MPDS. It proves to be particularly useful in the objective assessment of treatment results. It has also been proven to be useful for diagnosis of tender spots and trigger points.

Fischer (1986) states that pressure threshold is the minimum pressure that induces pain or discomfort. The algometer (pressure threshold meter) is designed to measure threshold pressure tolerance and tissue compliance in relation to trigger points, and will thus provide objective data for the assessment of the patient's condition. Fischer (1987) performed a study on the pressure threshold measurement for diagnosis of trigger points and evaluation of treatment results. The findings were that the algometer was effective for documentation of treatment effects, along with reliability of results. "Changes in pressure threshold obtained under standard clinical conditions, can therefore be regarded as reliable data" (Fischer, 1987).

Symptoms reported by the patient should also change after trigger point therapy. The procedure of taking a pressure reading was as follows. The algometer was set to zero and then pressed precisely over the active trigger point up to the pressure threshold (minimum pressure causing pain or discomfort) of that patient. The reading, obtained in kilograms per square centimetre, indicated the sensitivity of the trigger points to pain. A reading was

taken on each active trigger point in the iliotibial band and the tensor fasciae latae muscle. The sum of the readings for each trigger point were divided by the number of trigger point readings taken. Thus a single average reading was provided for that patient at that particular consultation.

The more active the trigger point the more sensitive it was to pressure, thus a post-treatment decrease of trigger point sensitivity to pain indicated that the treatment was proving effective. The increase in pain threshold should have correlated with a decrease in the signs and symptoms experienced by the patient.

The statistics used to evaluate this data (subproblem two) was Wilcoxon's paired signed rank test for intra group analysis. The tests were performed using the computer software programme STATGRAPHICS PLUS VERSION 6 supplied by MANUGISTICS INC. The processed data was presented in tabular format for easy interpretation.

In the above cases the Wilcoxon's paired signed rank test was used because it is a powerful test (less restrictive, yet very near equivalence in sensitivity to the T test) for non-parametric data with small sample sizes (Daniel 1978: 31-36). This was ideal for the data in this study. Furthermore, the assumption of normally distributed data was also not of importance when applying this test.

The null hypothesis for the above data was, within each group there was no improvement of the patients with regard to the objective and subjective clinical features. The alternative hypothesis was that within each group there was significant improvement of the patient with regard to objective and subjective clinical features (Steyn et al., 1994 : 415-421).

Once the objective results of subproblem one and subproblem two were processed, the results of subproblem three, namely finding out which of the two treatment types was more effective, could be determined. This was achieved using the Mann-Whitney U Test at the 5% level of significance. This test was chosen for its application to an inter-group statistical analysis as well as being held in high regard to power-efficiency (Daniel, 1978 : 82-86). Furthermore, the assumption of normally distributed data is not of importance when applying this test.

The null hypothesis for subproblem three is that there was no significant difference in the effects of MFTPT as compared to placebo therapy in the treatment of IBFS. An alternate hypothesis is that MFTPT will produce significantly better results than detuned ultrasound in the treatment of active trigger points (Steyn et al. 1994 : 415-421).

CHAPTER FOUR

RESULTS

4.1 Demographic Data

Table 4.1.1 **Demographic Data : Patient Data**

	EXPERIMENTAL GROUP	CONTROL GROUP	TOTAL
Age Distribution			
20-30	7	10	17
30-40	6	4	10
older than 40	2	1	3
Average age	32.3 (Max. age:20) (Min. age:47)	28.4 (Max. age:20) (Min. age:44)	30,3 (Max. age:20) (Min. age:47)
Gender Distribution			
Females	5	5	10
Males	10	10	20
Racial Distribution			
White	12	14	26
Black	2	0	2
Indian	1	1	2

Table 4.1.2 Demographic Data - History

		EXPERIMENTAL GROUP	CONTROL GROUP	TOTAL
Side of injury				
Left Knee		7	10	17
Right Knee		5	4	9
Bilateral		3	1	4
Duration of Symptoms (weeks)				
< 2 weeks		0	2	2
2 - 4 weeks		12	12	24
> 4 weeks		3	1	4
Severity of pain on initial consultation				
Grade 1.	Pain after running, not restricting distance or speed	1	3	4
Grade 2.	Pain during running, not restricting distance or speed	5	5	10
Grade 3.	Pain during running, restricting distance and speed	7	7	14
Grade 4.	Pain is so severe, it prevents running	1	0	1
Grade. 5	Pain continuous, during daily activities	1	0	1
Previous running injuries				
Runner's Knee (Patello-Femoral Pain Syndrome)		0	1	1
Shinsplints (Posterior Tibial Syndrome)		0	2	2
Achilles Tendonitis				
None		2	0	2
		13	12	25
Other Practitioners Consulted for current injury				
- Chiropractor		0	2	2
- Physiotherapist		0	2	2
- Medical doctor		0	0	0
- None		15	11	26

Table 4.1.2 Demographic Data - History - continued

	EXPERIMENTAL GROUP	CONTROL GROUP	TOTAL
Mileage per week - eight weeks before injury (Kilometres)			
<40 km	5	6	11
40-80 km	4	8	12
80 - 120 km	4	1	5
>120km	2	0	2
Numbers of marathons and/or ultra marathons			
0	6	9	15
1	0	1	1
2-4	3	2	5
>4	6	3	9
Number of running years			
<1	0	0	0
1	7	6	13
2-4	5	4	9
>4	4	4	8
Running speciality			
Social	7	9	16
Competitive short distance	0	0	0
Competitive marathon/ultra marathon	8	6	14
Terrain run on			
Road	13	13	26
Dirt	0	0	0
Grass	1	1	2
Sand	0	0	0
Track	0	0	0
Treadmill	1	1	2

Table 4.1.2 Demographic Data - History - continued

	EXPERIMENTAL GROUP	CONTROL GROUP	TOTAL
Shoes			
Recently changed shoes	0	1	1
Worn out shoes	5	1	6
Hard shoes	3	3	6
Non faulty shoes	7	10	17
Training			
Long slow distance	9	7	16
Fast sustained effort	1	0	1
Interval training	5	8	13
Recent changes in training schedule			
Increased speed	4	5	9
Increased distance	10	9	19
Increased hill running	1	1	2
Time spent stretching per day (minutes)			
0	8	2	10
0-10	5	12	17
11-20	2	1	3

Table 4.1.3 Demographic Data - Physical examination

	EXPERIMENTAL GROUP	CONTROL GROUP	TOTAL
Body type			
Ectomorph	13	12	25
Mesomorph	1	2	3
Endomorph	1	1	2
Knee alignment			
Neutral	10	9	19
Genu Varum	3	6	9
Genu Valgum	2	0	2
Foot alignment			
Neutral	7	5	12
Supination	4	3	7
Pronation	4	7	11
Arch Structure			
Normal	8	13	21
Pes Planus	4	2	6
Pes Cavus	3	0	3
Noble's Compression Test			
Positive	14	11	25
Negative	1	4	5
Ober's Test			
Positive	7	1	8
Negative	8	14	22
Tensor Fasciae Latae Trigger Points			
Active	3	5	8
Latent	12	10	22
None	0	0	0

4.2 RESULTS OF SUBJECTIVE AND OBJECTIVE FINDINGS :

The remainder of this chapter covers the results obtained from the statistical analysis of data collected from the following measurement criteria :

- Algometer readings
- The Numerical Pain Rating Scale 101
- The McGill Pain Questionnaire

Please note that all measurements were taken before each treatment in that consultation.

The results obtained from the statistical analysis are tabulated to display the mean for each group as well as the exceedance probability value (p-value) which is compared to the level of significance set at 0.05 for all the tests.

It must be noted that 14% if the experimental group were 100% pain free before the completion of the tenth treatment. Seven percent were 100% pain free with four treatments. These patients then therefore received a follow up consultation one month after the fourth treatment. The remaining 7% were 100% pain free within six treatments. The follow up consultation was therefore scheduled one month after the sixth treatment.

Due to the fact that certain patients were 100% pain free before the completion of the ten treatments, this resulted in an unequal number of patients in the experimental and control group at certain consultations. For example, fifteen patients formed the control group and thirteen patients formed the experimental group at the tenth consultation. This unequal number between the groups resulted in certain data not being amenable to statistical analysis. This explains why only the data from the placebo is tabulated in the following tables : Table 4.3; table 4.4; table 4.6; table 4.7; table 4.9; table 4.10; table 4.12 and table 4.13.

4.2.1 Subproblem One

The subjective responses of the patients to treatment were recorded using the Numerical Pain Rating scale 101; McGill Pain Questionnaire and The Pain Disability Index. The following results were obtained:

Table 4.2 The mean values and results of the Wilcoxon's paired signed rank test for the Numerical Pain Rating Scale 101 readings of the two groups during the period between the initial consultation ie. first treatment (T1) and the sixth treatment (T6)

	T1	T6	P-Value
Placebo Group	38.33	28.50	0.0134283
Experimental group	43.73	23.17	0.0001504

The null hypothesis is rejected for both groups which indicates that at the 5% level of significance a statistically significant change took place between treatment one and treatment six.

Table 4.3 : The mean values and results or the Wilcoxon's paired signed gained rank test for the Numerical pain Rating scale 101 reading of the two groups during the period of the sixth treatment (T6) and the tenth treatment (T10)

	T6	T10	P-Value
Placebo Group	28.50	23.27	0.065285

The null hypothesis is accepted for the placebo group which indicates at the 5% level of significance no statistically significant change took place between treatment six and treatment ten.

Table 4.4 : The mean values and results of Wilcoxon's paired signed rank test for the Numerical Pain Rating Scale 101 readings of the two groups between the tenth treatment (T10) and the follow up consultation (FC)

	T10	FC	P-Value
Placebo Group	23.27	22.33	0.308536

The null hypothesis is accepted for the placebo group which indicates at the 5% level of significance no statistically significant change took place between treatment ten and the follow up consultation.

Table 4.5 : The mean values and results of Wilcoxon's paired sign rank test for the McGill Pain Questionnaire readings of the two groups during the period between the initial consultation ie. the first treatment (T1) and the sixth treatment (T6)

	T1	T6	P-Value
Placebo Group	25.00	18.93	0.0132501
Experimental group	18.87	7.47	0.0012845

The null hypothesis is rejected for both groups which indicates at the 5% level of significance a statistically significant change took place between treatment one and treatment six.

Table 4.6 : The mean values and results of Wilcoxon's paired signed rank test for the McGill Pain Questionnaire readings of the two groups during the period between the sixth treatment (T6) and the tenth treatment (T10)

	T6	T10	P-Value
Placebo Group	18.93	14.53	0.224845

The null hypothesis is accepted for the placebo group which indicates at the 5% level of significance no statistically significant change took place between treatment six and treatment ten.

Table 4.7 : The mean values and results of Wilcoxon's paired rank test for the McGill Pain Questionnaire readings of the two groups during the period between the tenth treatment (T10) and the follow up consultation (FC)

	T10	FC	P-Value
Placebo Group	14.53	11.13	0.0206133

The null hypothesis is rejected for the placebo group which indicates at 5% level of significance a statistically significant change took place between treatment ten and the follow up consultation.

Table 4.8 : The mean values and results of the Wilcoxon's paired signed rank test for the Pain Disability Index readings of the two groups between the initial consultation ie :the first treatment (T1) and the sixth treatment (T6)

	T1	T6	P-Value
Placebo Group	12.13	9.27	0.3759130
Experimental group	13.40	7.13	0.0002561

The null hypothesis is accepted for the placebo group which indicates at the 5% level of significance no statistically significant change took place during the period between the first treatment and the sixth treatment.

The null hypothesis is rejected for the experimental group which indicates at the 5% level of significance a statistically significant change took place during the period between the first treatment and the sixth treatment.

Table 4.9 : The mean values and results of the Wilcoxon's paired rank test for the Pain Disability Index readings for the two groups during the period between the sixth treatment (T6) and the tenth treatment (T10)

	T6	T10	P-Value
Placebo Group	9.27	8.53	0.341544

The null hypothesis is accepted for the placebo group which indicates at the 5% level of significance no significant change took place during the period between the sixth treatment and the tenth treatment.

Table 4.10 : The mean values and results of the Wilcoxon's paired rank test for the Pain Disability Index readings of the two groups during the period between the tenth treatment (T10) and the follow up consultation (FC)

	T10	FC	P-Value
Placebo Group	8.53	5.99	0.50

The null hypothesis is accepted for the placebo group which indicates at the 5% level of significance no statistically significant change took place during the first period between the tenth treatment and the follow up consultation.

4.2.2 Subproblem Two

The objective response of the patient to treatment was recorded using an algometer. The following results were obtained.

Table 4.11 The mean values and results of the Wilcoxon's paired signed rank test for the algometer readings of the two groups during the period between the initial consultation ie : first treatment (T1) and the sixth treatment (T6)

	T1	T6	P-Value
Placebo Group	5.59	5.54	0.3027870
Experimental group	4.01	6.63	0.0001504

The null hypothesis is accepted for the placebo group which indicates at the 5% level of significant no statistically significant change took place during the period between the first treatment and the sixth treatment.

The null hypothesis is rejected for the experimental group which indicates at the 5% level of significance a statistically significant change took place during the period between the first and the sixth treatment.

Table 4.12 : The mean values and results of the Wilcoxon's paired signed rank test for the algometer readings for the two groups during the period between the sixth treatment (T6) and the tenth treatment (T10)

	T6	T10	P-Value
Placebo Group	5.54	5.43	0.150849

The null hypothesis is accepted for the placebo group which indicates at the 5% level of the significance no statistically significant change took place during the period between the sixth treatment and the tenth treatment.

Table 4.13 : The mean values and results of Wilcoxon's paired signed rank test for the algometer readings of the two groups during the period between the tenth treatment (T10) and the follow up consultation (FC)

	T10	FC	P-Value
Placebo Group	5.43	5.11	0.302787

The null hypothesis for the placebo group is accepted which indicates at the 5% level of significance no statistically significant change took place during the period between the tenth treatment and the follow up consultation.

4.2.3 Subproblem Three

The results of the above subproblems have now been combined in order to determine whether the experimental group (ie: needling, cryotherapy and stretch) performed significantly better than the placebo group. The following results were obtained:

Table 4.14 : The mean values and results of the Mann-Whitney U test comparing the Numerical Pain Rating Scale 101 readings of both groups (experimental (E) and placebo (P)) firstly at the initial consultation and at the sixth treatment

	P	E	P-Value
Initial consultation ie ; treatment one	11.47	19.53	0.0059544
Treatment six	16.90	14.10	0.1965215

The null hypothesis is rejected for the first treatment which indicates that at the 5% level of significance there is a statistically significant difference between the two groups.
The null hypothesis is accepted for the sixth treatment which indicates at the 5% level of significance there is no statistically significant difference between the two groups.

Table 4.15 The mean values and results of the Mann-Whitney U test comparing the Numerical Pain Rating scale 101 readings of both groups (experimental (E) and placebo (P) firstly at the sixth treatment and at the tenth treatment.

	P	E	P-Value
Treatment six	16.90	14.10	0.1965215
Treatment ten	16.77	11.88	0.0609310

The null hypothesis is accepted for both the sixth and tenth treatments which indicates at the 5% level of significance there is no statistically significant difference between the two groups.

Table 4:16 : The mean values and results of the Mann-Whitney U Test comparing the Numerical Pain Rating Scale 101 of both groups (experimental (E) and placebo (P) at the tenth treatment and the follow up consultation

	P	E	P-Value
Treatment ten	16.77	11.88	0.0609310
Experimental group	16.37	9.59	0.0132020

The null hypothesis is accepted for the tenth treatment which indicates at the 5% level of significance there is no statistically significant difference between the two groups.

The null hypothesis is rejected for the follow up consultation which indicates at the 5% level of significance there is a statistically significant difference between the two groups.

Table 4.17 : The mean values and results of the Mann-Whitney U Test comparing the McGill Pain Questionnaire readings of both groups (experimental (E) and placebo (P)) firstly at the initial consultation and at the sixth treatment

	P	E	P-Value
Initial Consultation ie : Treatment one	16.93	14.07	0.1908290
Treatment six	19.23	11.77	0.0098208

The null hypothesis is accepted for the first treatment which indicates that the 5% level of significance there is no statistically significant difference between the two groups.

The null hypothesis is rejected for the sixth treatment which indicates that at the 5% level of significance there is a statistically significant difference between the two groups.

Table 4.18 : The mean values and results of the Mann-Whitney U Test comparing the McGill Pain Questionnaire readings of both groups (experimental (E) and placebo (P)) at the sixth treatment and the tenth treatment.

	P	E	P-Value
Treatment six	19.23	11.77	0.0098208
Treatment ten	18.30	10.12	0.0037792

The null hypothesis is rejected for both the sixth and tenth treatment which indicates at the 5% level of significance there is a statistically significant difference between the two groups.

Table 4.19 : The mean values and results of the Mann-Whitney U Test comparing the McGill Pain Questionnaire readings of both groups (experimental (E) and placebo (P)) at the tenth treatment and at the follow up consultation

	P	E	P-Value
Tenth treatment	18.30	10.12	0.0037792
Follow up consultation	17.33	9.38	0.0062894

The null hypothesis is rejected for both the tenth treatment and the follow up consultation which indicates at the 5% level of significance there is a statistically significant difference between the two groups.

Table 4.20 : The mean values and results of the Mann- Whitney U test comparing the Pain Disability Index readings of both groups (experimental (E) and placebo (P)) firstly at the initial consultation and at the sixth treatment.

	P	E	P-Value
Initial consultation i.e. Treatment one	14.50	16.50	0.2725395
Treatment six	18.07	12.93	0.0554275

The null hypothesis is accepted for both groups which indicates at the 5% level of significance, there is no statistically significant difference between the placebo group and the experimental group

Table 4.21 : The mean values and results of the Mann-Whitney U test comparing the Pain Disability Index readings of both groups (experimental (E) and placebo (P)) at the sixth treatment and at the tenth treatment.

	P	E	P-Value
Treatment six	18.07	12.93	0.0554275
Treatment ten	17.2	11.38	0.0308194

The null hypothesis is accepted for the sixth treatment which indicates at the 5% level of significance there is no statistically significant difference between the two groups.

The null hypothesis is rejected for the tenth treatment which indicates at the 5% level of significance, there is a statistically significant difference between the two groups.

Table 4.22 : The mean values and results of the Mann-Whitney U test comparing the Pain Disability Index readings of both groups (experimental (E) and the placebo (P)) at the tenth treatment and at the follow up consultation

	P	E	P-Value
Treatment ten	17.20	11.38	0.0308194
Follow up consultation	15.77	10.41	0.0381990

The null hypothesis is rejected for the tenth treatment and the follow up consultation which indicates the 5% level of significance there is a statistically significant difference between the two groups.

Table 4.23 : The mean values and results of the Mann-Whitney U test comparing the algometer readings of both groups (experimental (E) and placebo (P)) firstly at the initial consultation and at the sixth treatment.

	P	E	P-Value
Initial consultation i.e. Treatment one	19.30	11.70	0.0095449
Treatment six	12.47	18.53	0.0309847

The null hypothesis is rejected for both groups which indicates at the 5% level of significance there is a significant difference between the experimental and placebo group.

Table 4.24 : The mean values and results of the Mann-Whitney U Test comparing the algometer readings of both groups (experimental (E) and placebo (P)) at the sixth treatment and at the tenth treatment

	P	E	P-Value
Treatment six	12.47	19.53	0.0309847
Treatment ten	9.337	20.46	0.0001949

The null hypothesis is rejected for both groups which indicates at the 5% level of significance there is an significant difference between the experimental and placebo group.

Table 4.25 : The mean values and results of the Mann-Whitney U test comparing the algometer readings of both groups (experimental (E) and placebo (P)) at the tenth treatment and the follow up consultation

	P	E	P-Value
Treatment ten	19.30	11.70	0,0095449
Follow up consultation	8.07	20.91	0.0001310

The null hypothesis is rejected for both groups which indicates at the 5% level of significance, there is a significant difference between the experimental and placebo group.

CHAPTER FIVE

DISCUSSION

5.1 DISCUSSION

Firstly, it is recognised that the selected sample of thirty patients is too small from which to draw statistically significant conclusions. Yet, the results of my study can be compared to the studies reported by Sutker et al. (1985) of forty eight patients as well as the study by Lindenberg et al. (1984) of thirty six patients.

Of the sixty patients who applied for the research, only forty five qualified to take part and of these only thirty completed the full course of treatments. Fifteen patients did not complete the full course due to the following reasons:

i) Three patients changed shoes during the treatment programme from hard shoes to soft shoes and therefore, although they continued to receive treatment, the data obtained from these patients did not contribute to the statistics. This is due to the fact that changing shoes could affect the results of treatment. Although during the treatment period the patients were required to run to the point when they first felt slight discomfort, they could not be expected to run in old, hard shoes as this was considered unethical. Thus, if the patient started running in new shoes, the data obtained was not utilized to furnish the statistics.

ii) Three patients who were allocated to the experimental group responded with a syncopal reaction when first needled and thus were discarded from the study. All three sweated excessively and felt as if they wanted to faint as well as complaining of nausea. These patients continued to receive treatment for IBFS in the form of cross friction, ultra sound, cold packs and stretching but therefore did not contribute to the results of the study. It is interesting to note that all three were fair skinned with light hair and blue eyes. This is in accordance with the observation made in the study by Sola (1984).

iii) The remaining 'drop-out` was presumed to be as a result of several factors, as

follows:

- 'Drop-out' from the placebo group were due to patients perceived lack of improvement.
- In both groups, the remainder of patient 'drop-outs' were as a result of non-compliance.

The reasons for non-compliance included transport problems, work/time constraints and discouragement/misinformation from friends.

To ensure that the data satisfied all the pre-set criteria, all pain questionnaires had to be fully completed under supervision of the researcher, and all algometer readings taken by the researcher. If the data, at any time, did not satisfy these criteria, the patient, along with the results were dismissed from the study. No data was excluded for these reasons.

Although IBFS has been observed in runners (Noble, 1979; Noble, 1980; Sutker et al. 1981; Lindenberg et al. 1984; Sutker et al. 1985; Jones and James, 1987:280 and Henderson, 1989) skaters; cross-country skiers (Orava, 1978; Noble 1979; and Noble 1985:32); cyclists (Noble, 1985;32 and Holmes et al. 1993) and football players (Martens et al. 1989), all of the sample group were runners.

5.2 FROM THE DEMOGRAPHICAL DATA

The demographical data indicates that IBFS is most common in the twenty to thirty year old age distribution, with the average age being thirty years old. This is in accordance with Sutker et al. (1981) and Sutker et al. (1985) who found the average age to be thirty one years old, and Lindenberg et al. (1984) who found that runners most commonly affected with IBFS were between the ages of twenty to forty years old.

Of the thirty patients with IBFS, twenty were male. This predominance of males is in accordance with Sutker et al. (1981); Lindenberg et al. (1984) and Sutker et al. (1985).

The present study showed that 87% of the sample size were White and the remainder showed equal distribution of Blacks and Indians. This is possibly an unrealistic sample as advertising that was undertaken to acquire patients for the study was in the form of leaflets sent per "Rave Sports Promotions". The majority of the clients of this organization are European in origin and therefore these are probably deceiving statistics.

The present findings concerning the side of the injury differ from those of Lindenberg et al. (1984) and Firer (1989). In my study 57% of the sample had left sided injuries and 30% had right sided injuries. The remainder were affected bilaterally. In contrast, Firer (1989) and Lindenberg et al. (1984) state that right-sided IBFS is dominant. This is rationalized due to the fact that both Firer (1989) an Lindenberg et al. (1984) performed their studies in South Africa where right-hand drive occurs. The runner thus runs on the right hand side of the road facing the oncoming traffic (due to safety reasons), and therefore develops a right hand sided injury. Yet, in this study, also performed in South Africa, left sided injuries were more prevalent.

This study also showed that 100% of runners presenting with a left sided injury, claimed to run on the left hand side of the road and 100% of runners using the right hand side of the road developed right sided injuries. Thus, 100% of runners who favoured the left or right hand side of the road sustained injuries on the down slope of the cambre. This is in accordance with Noble (1979); Lindenberg et al. (1984) Noakes (1992:451); Sutker et al. (1985); Firer (1989); Noakes (1992:451) and Reid (1992:424) who state that the injury occurs on the side of the body corresponding to the side of the road the runner runs.

Concerning runners most susceptible to IBFS the present study differs from those of Orava (1978); Noble (1979) and Lindenberg et al. (1984). This study demonstrates that the incidence of IBFS is not related to the number of marathons and/or ultra marathons run nor the mileage per week. In contrast, Lindenberg et al.(1984) found that the runners prone to this injury have run at least one standard marathon and train more than forty kilometres per week. The current findings were similar to those of Sutker et al.(1981) and Sutker et al.(1985) in that most runners with IBFS had been running for more than one year.

The data indicates that 80% of the sample showed symptoms for four weeks or less. Also, the majority of patients presented with a grade two or grade three injury (see Appendix 13). Both these findings are in accordance with Lindenberg et al.(1984).

Only 17% had previous injuries, these being Runners Knee (Patello-Femoral Pain Syndrome); Shinsplints (Posterior Tibial Syndrome) and Achilles Tendonitis. These findings are remarkably similar to those of Lindenberg et al.(1984). Also, 87% did not consult other practitioners prior to the first consultation for IBFS. Those who did, consulted Physiotherapists or Chiropractors and treatment ranged from ultrasound, electrical stimulation, icing, manipulation, local heat application and massage.

According to Noble (1979); Noble (1980); Noble (1985:32); Lindenberg et al.(1984); and Sutker et al. (1985), IBFS is predominantly seen in long distance runners. Although 100% of the sample size were long distance runners, only 47% of these were competitive marathon or ultra marathon runners, with the remainder being social runners. Yet 57% of the sample did long slow distance training whilst 43% performed interval training. One athlete of the thirty performed fast sustained effort training.

Eighty seven percent of the sample in this study ran on tarmac roads. These results are similar to those of Sutker et al. (1981); Lindenberg et al. (1984) and Sutker et al.(1985). The remainder ran on grass and on the treadmill.

Twenty percent of runners of the current study presented with 'worn-out` shoes and 20% presented with hard running shoes on initial consultation. These findings are remarkably similar to those of Sutker et al. (1981) and Sutker et al.(1985), where 16% of patients presented with "worn out" shoes on initial consultation. In this study, "worn out" shoes indicated generalized smoothness of the sole of the shoe and compression of the midsole along the angle of the heel flare. All 20% of runners who were presented with 'worn-out` shoes had more than one millimetre of wear on the posterior lateral edge of the heel. Noble(1980) states that in his study 47% of the patients showed more than one millimetre of wear on the posterior lateral edge of heel when they first presented with iliotibial band pain. Lindenberg et al.(1984) states that runners are more susceptible to IBFS if they run in a hard shoe. Also, of the thirty runners in my sample group, one presented with recently changed shoes. This findings is supported by Baker(1995) who states that shoe changes lead to inadequate shock absorption and thus predispose IBFS.

Changes to the training schedule prior to the initial consultation included increased speed, increased distance and increased hill running. This is in agreement with Noble (1979); Noble (1980); Sutker et al. (1981), Sutker et al. (1985) Noakes and Granger (1990:97-98) and Noakes (1992:450-451). In the present study the majority of the sample (63%) increased their running distance.

Fifty seven percent of the sample in this study stretched for less than ten minutes per day. A third of the sample did no stretching. These results are remarkably similar to those of Lindenberg et al.(1984).

It was revealed on physical examination in this study that 93% of the sample were either ectomorph or mesomorph body type. This is substantiated by Sutker et al.(1981) and Sutker et al.(1985) who state that IBFS is rarely seen in endomorphs probably due to the fact that they do not run as far, as well as fat in the tissue around the knee may serve as lubrication under the tight iliotibial band.

Concerning knee alignment, the majority of patients (93%) in this study showed neutral or varum alignment. In varum alignment, gaps between knees varied from three to ten centimetres as they did in the Noble(1980) study. The predominance of neutral or varum alignment are confirmed by studies performed by Sutker et al. (1981); Lindenberg et al. (1984); Sutker et al. (1985); Martens et al. (1989); Reid (1992:425) and Baker (1995). Noble et al. (1982); McBryde et al. (1985:410) and James (1987:282), state that genu varum and leg length discrepancies cause tightness of the iliotibial band which predisposes IBFS. In the present study, however, no leg length discrepancies were observed. Lindenberg et al.(1984) reported that 56% of his sample group had leg length inequality, yet the side of the discrepancy did not correlate with the side of the injury.

Twenty three percent of my sample showed inadequate subtalar joint pronation and 37% showed excessive subtalar pronation. Lindenberg et al.(1984) state that both excessive and inadequate ankle joint pronation can contribute to IBFS.

According to Sutker et al. (1981); Sutker et al. (1985), Noakes and Granger (1990:99) and Noakes (1992:450); high arched rigid feet are unable to absorb shock adequately which is transferred to the iliotibial band and therefore causing injury. Yet, according to Noakes and Granger(1990:99) some runners with extremely flat feet which are also unable to absorb shock adequately may be equally at risk. The current findings concerning arch structure showed that 20% had pes planus and 10% had pes cavus, thus possibly leading to IBFS. None of

those sampled utilized orthotics.

Noble's Compression Test was positive in 83% of my sample. According to Noakes and Granger(1990:97), Noble's Compression Test may not elicit pain if the runner has not run for the previous three or more days. Due to this reason, the Noble Compression Test was not an essential sign for the patient to be accepted into the research, yet it did confirm the diagnosis. If Noble's Compression Test was negative, the diagnosis was based on the history. Also, it was essential for the patient to have pain over the lateral knee and tenderness over the lateral femoral epicondyle for two to four centimetres to be accepted into the study. In five of the thirty patients, Noble's Compression Test was negative and all five had not run for three days prior to the initial consultation.

The current study showed that Ober's Test was positive in 26% of cases and negative in the remainder. In my findings Ober's test did not reveal subtle tightness and was only positive in extreme cases.

In 100% of cases, trigger points were found in the iliotibial band and tensor fasciae latae of the involved leg. At least five active trigger points were found in the iliotibial band of the involved leg on initial consultation. Only 26% of the trigger points in the tensor fasciae latae muscle were active, the remainder being latent. Latent trigger points were not needled.

Although, at least five active trigger points were found in the iliotibial band on initial consultation, they were noted to decrease in number in patients in the experimental group as they responded to treatment. Therefore, on initial consultation approximately five active trigger points were needled. Yet, as the patient responded to treatment less trigger points were found and therefore less trigger points were needled. Thus, possibly on the final consultation only one

trigger point was needled.

An observation was made that trigger points increased in severity from the proximal iliotibial band to the distal iliotibial band. That is, the patients stated that needling trigger points in the distal iliotibial band were far more painful than needling trigger points in the proximal iliotibial band, with those trigger points closest to the lateral femoral epicondyle being the most severe.

5.3 DISCUSSION OF THE RESULTS

During the period between the sixth treatments and the follow up consultations only thirteen of the fifteen patients in the experimental group received the tenth treatment and eleven of the same fifteen attended the follow up consultation. This is due to the fact that these patients were 100% improved before the completion of the tenth treatment. Once patients considered themselves to have reached 100% improvement and could run at a pre-injury symptom-free level, they were required to have two more treatments. Yet, this did not necessarily include the tenth treatment and the follow up consultation. Therefore, due to the fact that only thirteen patients attended the tenth treatment and eleven patients attended the follow up consultation, there were unequal sets of data which could not be statistically analyzed. Thus, the Numerical Pain Rating Scale 101, McGill Pain Questionnaire, Pain Disability Index or algometer readings could not be statistically analyzed for the experimental group between the sixth treatment and the follow up consultation (See table 4.3; 4.4; 4.6; 4.7; 4.9; 4.10; 4.12; 4.13). Yet, it must be emphasized that 27% of the experimental group were 100% better before completion of the allotted follow up consultation. This indicates perceived improvement in patients forming part of the experimental group.

It must be noted that there was no relationship between the duration of the patient's symptoms on initial consultation and speed of recovery.

5.3.1 ANSWERS TO SUBPROBLEM ONE

Wilcoxon's paired signed rank test for the Numerical Pain Rating Scale 101 readings during the period between the first treatment and the sixth treatment (table 4.2) resulted in rejection of the null hypothesis for both groups, indicating that a significant change occurred during this period. This change was in the form of reduction of the mean scores over the specified time, indicating that the patients perceived a decrease in pain intensity as the treatment took effect.

Yet, during the period of the sixth treatment and tenth treatment as well as the follow up period (table 4.3 and table 4.4) the null hypothesis for the placebo group is accepted which indicates that no statistically significant change took place during this period. Thus, patients in the placebo group maintained perceived improvement of their condition during the period between the sixth treatment and the follow up period but did not improve any further.

Wilcoxon's paired signed rank test for the McGill Pain Questionnaire readings during the period between the first treatment and the sixth treatment (table 4.5) resulted in rejection of the null hypothesis for both groups, indicating that the patients perceived a decrease in aspects pertaining to pain as the treatment took effect.

During the period of the sixth to tenth treatment, Wilcoxon's paired signed rank test (table 4.6) resulted in acceptance of the null hypothesis for the placebo group, which indicates no statistically significant change took place during this period. Yet table 4.7 shows that during the period from the tenth treatment to the follow up consultation, the null hypothesis for the placebo group is rejected indicating a statistically significant change took place.

Therefore, it was determined that there was significant improvement in the placebo group between the period of the first treatment and the sixth treatment, which was maintained from the sixth to the tenth treatment. Over the follow up period, a significant improvement was again seen in the placebo group indicated by a reduction in mean scores and thus

reduction of pain.

Wilcoxon's paired signed rank test for the Pain Disability Index reading during the period between the first treatment and the sixth treatment (table 4.8) resulted in rejection of the null hypothesis for the experimental group, indicating that a statistically significant change took place during this period. This change was in the form of reduction of the mean scores over the specified time, indicating that the patients perceived a reduction in pain and showed improvement of their condition as treatment took effect. During the period between the sixth treatment and the follow up consultation the data for the experimental group could not be statistically analyzed.

Wilcoxon's paired signed rank test for the Pain Disability Index readings during the period between the first treatment and the follow up consultation (table 4.8; 4.9; 4.10) resulted in the acceptance of the null hypothesis for the placebo group, indicating that no statistically significant change occurred during the entire treatment and follow up period. Thus, patients forming part of the placebo group did not respond to treatment.

The subjective tests showed similar results. Both the Numerical Pain Rating Scale 101 and McGill Pain Questionnaire showed rejection of the null hypothesis for both groups during the period between the first treatment and the sixth treatment. During this same period, the Pain Disability Index, however only indicated rejection of the null hypothesis for the experimental group and acceptance of the null hypothesis for the placebo group. All three subjective tests indicated acceptance of the null hypothesis for the placebo group during the period between the sixth and tenth treatment. During the follow up period only the McGill Pain Questionnaire indicated rejection of the null hypothesis for the placebo group.

Therefore, the subjective tests indicate that a generally significant improvement with regards to the pain perception of the problem is shown in both groups during the period between the first and sixth treatment and no significant improvement was seen for the

placebo group between the sixth treatment and the follow up consultation.

The fact that the placebo group showed significant improvement between the first and the sixth treatment was not expected as the placebo treatment was not supposed to be effective. This can be explained due to the fact that 30% of placebo treatments will be effective (Broome, 1994). The experimental group improved as a result of the applied treatment.

A possible reason for the placebo group lack of improvement after the sixth treatment may be due to the fact that this group did not receive patient education or prescribed specific stretch exercises. During the follow up period, although the experimental group data could not be statistically analyzed due to unequal sample sizes, it was observed that a general improvement was seen. This can be explained as the nature of treatment for the experimental group included patient education and stretch exercises which would continue to influence the patients improvement even after the active treatment had stopped.

These subjective results support hypothesis one, which states that the experimental group will show significant improvement in the subjective presentation of the patient.

5.3.2 ANSWERS TO SUBPROBLEM TWO

Wilcoxon's paired signed rank test for the algometer readings during the period between the first treatment and the follow up consultation (table 4.11; 4.12; 4.13) resulted in acceptance of the null hypothesis for the placebo group, indicating that no statistically significant change occurred during the entire treatment and follow up period. Thus patients forming part of the placebo group did not respond to treatment.

Wilcoxon's paired signed rank test for the algometer readings during the period between the first treatment and the sixth treatment (table 4.11) resulted in rejection of the null hypothesis for the experimental group indicating that a statistically significant change took

place during this period. Pressure to pain tolerance of the experimental group did increase. During the period between the sixth and the follow up consultation the data for the experimental group could not be statistically analyzed due to unequal sample sizes.

These objective findings support hypothesis two, which states that the experimental group will show significant improvement in the objective presentation of the patient.

It is theorized that the unexpected improvement in the Numerical Pain Rating Scale 101 and McGill Pain Questionnaire of the placebo group was a subjective response and that the objective results would not support the above findings. This theory was substantiated when the algometer readings showed that no significant change occurred during the entire treatment and follow up period and thus patients forming part of the placebo group did not respond to treatment (tables 4.2; 4.5; 4.11; 4.12; 4.13).

Due to the fact that during the period between the sixth treatment and the follow up consultations the data of the experimental group could not be statistically analyzed, adds difficulty in determining whether improvement occurred. Also difficult to establish is whether or not those patients who continued treatment (i.e. those patients not 100% improved prior to completion of the tenth treatment) were compliant with regard to stretching programmes and whether or not they avoided exposure to the original or new precipitating factors. Yet, it can be concluded from observation that improvement was perceived and that patients were generally compliant.

5.3.3 ANSWERS TO SUBPROBLEM THREE

The results from the subjective findings were compared using the Mann-Whitney U test and it was found that results from the three subjective tests varied tremendously (tables 4.14; 4.15; 4.16; 4.17; 4.18; 4.19; 4.20; 4.21; 4.22). Thus no conclusions may be specifically drawn.

Table 4.14. shows that when utilizing the Mann-Whitney U test comparing the Numerical Pain Rating Scale 101 for both groups, a statistically significant difference was seen between the two groups on initial consultation. This shows that the experimental group reported greater pain than the placebo group on initial consultation.

The subjective results demonstrated that the experimental group showed no greater positive results than the placebo group in the following circumstances:

- When the Numerical Pain Rating Scale 101 was utilized at the first treatment and sixth treatment.
- The McGill Pain Questionnaire showed no difference between the two groups at the first treatment.
- The Pain Disability Index also showed that the experimental group showed no greater positive results than the placebo group at the first treatment and sixth treatment.

These unexpected results are explained due to the fact that 30% of placebo treatments will be effective (Broome, 1994).

Again it was theorized that the unexpected improvement in the placebo group was a subjective response and that the objective results would not support the above findings. This theory was substantiated when the results from the objective findings were compared using the Mann-Whitney U test and it was found that the experimental group out-performed the placebo group during the

period from the first treatment to the follow up consultation (table 4.23; 4.24; 4.25). Thus there was a significant difference between the two groups during this period and this supports hypothesis three which states that the experimental group will prove to produce better results when compared to the placebo group.

The experimental group improved more than the placebo group in both the subjective and objective measurements. Therefore, this strongly suggests the hypotheses that MFTPT is effective in the treatment of IBFS, correct.

CHAPTER SIX

CONCLUSION &

RECOMMENDATIONS

6.1 CONCLUSION

When the results of both groups were combined it was found that the group receiving the authentic MFTPT improved more than the placebo group in both the subjective and objective measurements and in doing so supported all the stated hypotheses correct. It can thus be stated that MFTPT, including stretching exercises and patient education, is an effective form of treatment for IBFS.

6.2 RECOMMENDATIONS

This study should be repeated using a larger sample size so that more accurate conclusions may be derived. Should the study be repeated it is recommended that allowances be made for the patient and it is advised that a set number of treatments per week not be applied. Rather, it is suggested that the patient make a set number of consultations within a set period that is more suitable to them. The period should be long enough for this to occur. It would be of interest to conduct a follow-up study at six months, one year and two years, following the last treatment to establish how effective the treatment might be over a longer period and how many of the patients have maintained their exercise routine.

With regard to maintenance programmes, I would monitor the patients while performing specific stretching regimes under my supervision after each treatment. I would schedule consultations twice a week during the follow-up period to observe the stretch exercises being performed. This would serve to remove patient non-compliance. Most literature dealing with myofascial pain stresses the importance of stretching the affected muscles, while Lewit and Simons (1984) demonstrated that long term relief was elicited in 63% of myofascial patients using stretching techniques. Thus it should be an objective of the study that all patients comply with their stretching programmes to ensure best results.

Forty seven percent of the experimental group complained of post needle soreness after

treatment. An observation was made from the history that these patients had a low intake of Vitamin B and ascorbic acid due to the fact that they patients did not include fruit and vegetables in their diet. Travell and Simon (1983:65) link this with post needle soreness. These patients were therefore advised to increase their vitamin B and ascorbic acid intake.

Alignment of the foot was observed and not measured. In the study performed by Lindenberg et al.(1984), 83% of the patients with IBFS showed varum alignment of the foot (both hindfoot and forefoot), Therefore, this should definitely be taken into account in future studies. Noble (1980) measured heel valgus or varus with aid of a goniometer.

It was established from the current study that 57% of the patients examined had left sided injuries. This group were noted to be left-sided road runners. Therefore, the runners ran with their backs to the traffic. The reason must be established why these runners engaged themselves in such a potentially dangerous situation. It is essential to educate runners to switch sides of the road to prevent IBFS, but whenever running on the left hand side of the road to be alert for safety reasons.

6.3. CONSIDERATIONS FOR FURTHER INVESTIGATIONS

It was noted in the current study that needling trigger points in the distal iliotibial band was more painful than needling trigger points in the proximal iliotibial band, with those being closest to the lateral femoral epicondyle being the most severe. Further research should be undertaken to establish reasons for this.

The present study showed that when utilizing the Mann-Whitney U Test comparing the Numerical Pain Rating Scale 101 for both groups, a statistically significant difference was observed between the two groups on initial consultation (Table 4.14). This indicates that the experimental group reported greater pain than placebo group on initial consultation. A similar study should be repeated, in order to ascertain more accurate conclusions.

It is recommended that two separate pain questionnaires be designed, one for the treatment sessions and one for the follow up consultations. This is due to the fact that patients tended to refer to the pain they had before and during the treatment rather than the pain they experienced subsequent to the last treatment.

It would be interesting to note the outcome of treatment if latent trigger points in the tensor fasciae latae muscle were needled.

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APPENDICES

APPENDIX 1

[illegible]

[illegible]

Patient: _____ Date 0 _____

Pilo 0: _____

X-ray 0: _____

Age: _____ Sex: _____ Occupation: _____

Intern: _____ Signature: _____

FOR CLINICIAN'S USE ONLY

Initial visit clinician: Signature:

Case History:

Examination:
Previous: N
Other
Current: N
Other

X-ray Studies:
Previous: N
Other
Current: N
Other

Clinical path. lab.:
Previous: N
Other
Current: N
Other

Case review:
PIT: Conditional: Signed off: Final sign out:

Recommendations:

Patient's own history

1. Source of history:

2. Chief complaint: (patient's own words)

3. Present illness:

Location

Onset

Duration

Frequency

Pain (character)

Progression

Aggravating factors

Relieving factors

Associated S & S

Previous occurrences

Past treatment and outcome

4. Other complaints:

5. Past history:

General health review

Childhood illnesses

Adult illnesses

Psychiatric illnesses

Accidents/injuries

Surgery

Hospitalizations

6. Current health status and life-style:

Allergies

Immunizations

Screening tests

Environmental hazards
(home, school, work)

Safety measures
(seat belts, condoms)

Exercise and leisure

Sleep patterns

Diet

Current medication

Tobacco

Alcohol

Social drugs

7. Family history:

Immediate family:

Age
Health
Cause of death
DM
Heart disease
TB
HBP
Stroke
Kidney disease
CA
Arthritis
Anemia
Headaches
Thyroid disease
Epilepsy
Mental illness
Alcoholism
Drug addiction
Other

8. Psychosocial history:

Home situation

Daily life

Important experiences

Religious beliefs

9. Review of systems:

General

Skin

Head

Eyes

Ears

Nose/sinuses

Mouth/throat

Neck

Breasts

Respiratory

Cardiac

Gastro-intestinal

Urinary

Genital

Vascular

Musculoskeletal

Neurologic

Hematologic

Endocrine

Psychiatric.

[illegible]

Underline abnormal findings in RED and elaborate on back of relevant page, if necessary.
Mark "WNL" if normal.

Patient: _____ File #_____

Last name First name

Clinician: _____ Signature: _____

Intern: _____ Signature: _____

Date: _____

Weight: _____ Height: _____ Temp: _____

Ortho: Heart: _____ Pulse: _____ Respirations: _____

Blood pressure: Arm: L / R /

Legs: L / R /

General appearance:

GAINING EXAMINATION.

Miner's sign
Skin changes
Posture
  erect
  Adm's
Range of motion:

T/L spine: Flexion: 90 Fingers to floor
Extension: 30
R.lat.flex.: 30 Fingers down leg
L.lat.flex.: 30 Fingers down leg
Rot.to R.: 35
Rot.to L.: 35

Flex.

L.Rot. R.Rot.

L.lat flex. R.lat. flex.

Ext.

/ = pain-free limitation; // = painful limitation.

Romberg's sign.
Pronator drift.
Trendelenburg's sign.
Gait.
  rhythm
  balance
  pendulousness
  on toes
  on heels
  tandem
Half squat.
Scapular winging.
Muscle tone.
Spasticity/Rigidity:

Shoulder:
  skin
  symmetry
  ROM - glenohumeral
        scapulo-thoracic
        acromioclavicular
        elbow
        wrist
Chest movement
  inspiration
  expiration
Visual acuity

Breast examination:
  Inspection:
    skin
    size
    contour
    nipple
    any overhang
    hands against hips
    leaning forward.
  Palpation:
    axillary lymph nodes.

SPINAL EXAMINATION.

Spinal posture
Head
  scalp
  skull
  face
  skin
Eyes
  conjunctiva
  sclera
  cornea
  eyelids
  lacrimal gland
  nasolacrimal duct
  oligomat
  corneal reflex
  ocular movement

	R			L	
VI	V	III	VI	V	III

  visual fields
  accomodation
  iris
  pupils
  red reflex
  optic disc

vessels
general background
macula
vitreous
lens

Eyes:
  eyelids
  eye canal
  eyes
  auditory acuity
  Weber test
  Rinne test

Nose:
  external
  internal
    septum
    turbinates
    olfaction
  Sinuses (frontal & maxillary):
    tenderness
    transillumination
  Mouth and pharynx:
    lips
    buccal mucosa
    gums and teeth
    roof
    tongue
      inspection
      movement
      taste
      palpation
    pharynx
      inspection
      El X
  Neck:
    posture
    size
    swelling
    scars
    discoloration
    hair line

ROM:

Flexion:	45	chin to larynx
		chin to sternum
Extension:	55	forehead parallel to floor
L.lat.flex:	40	
R.lat.flex:	40	
L.rot.:	70	
R.rot.:	70	

Flex.

L.lat. R.lat.

L.Lat. flex. R.lat. flex.

Ext.

lymph nodes
trachea
thyroid
carotid arteries (thrills, bruit)
☐ V
☐ VII
☐ VIII (eyeball)
☐ IX
☐ XI
☐ –
inspection
ROM
deviation
Palpation
crepitus
tenderness

Neurological:
  Reflexes
    C5
    C6
    C7
    C8
    T1
  Tendon reflexes
    biceps
    triceps
    brachioradialis
  Muscle strength
    C5
    C6
    C7
    C8
    T1
  Coordination:
    point-to-point
    dysdiadochokinesis
Thorax:
  Chest:
    Inspection:
      skin
      shape
      respiratory distress
      rhythm (respiratory)
      depth "
      effort "
      intercostal/supraclavicular retraction
    Palpation:
      tenderness
      mass
      respiratory expansion
      tactile fremitus
    Percussion:
      lungs (posterior)
      diaphragmatic excursion
      kidney punch
    Auscultation:
      breath sounds
        vesicular
        bronchial
      adventitious sounds
        crackles (rales)
        rhonchi (rhonchi)
      voice sounds
        bronchophony
        whispered pectoriloquy
        egophony

Cardiovascular:
auscultation (aortic [illegible])
Allen's test

SPINAL EXAMINATION

JVP
HJ
auscultation heart (L.lat.[illegible])
respiratory excursion
percussion chest (anterior)
thrust palpation

The abdomen:
Inspection:
skin
umbilicus
contour
peristalsis
pulsations
hernias (umbilical/incisional)
Auscultation:
bowel sounds
bruit
Percussion:
general
liver
spleen
Palpation:
superficial reflexes
cough
light
rebound tenderness
deep
liver
spleen
kidneys
aorta
iatro-/retro-abdominal wall mass
shifting dullness
fluid wave
Acute abdomen:
where pain began and now
cough
tenderness
guarding/rigidity
rebound tenderness
Rovsing's sign
psoas sign
obturator sign
cutaneous hyperesthesia
rectal exam
Murphy's sign.

Male genitalia and hernias.

Inspection:
skin
prepuce
glans
meatus
skin/lies
scrotum
inguinal/femoral bulges

Palpation:
penis (tenderness/induration)
testes
epididymis
inguinal canal
femoral canal
external orifice reflux

Auscultation:
scrotal mass.

Peripheral vascular:

Inspection:
skin
nail beds
pigmentation
hair loss

Palpation:
pulses - radial, brachial, femoral, popliteal, post.tibial, dorsalis pedis
lymph nodes - epitrochlear, femoral (horizontal & vertical)
temperature (foot & legs)
Manual compression test
Retrograde filling (Trendelenburg) test
Arterial insufficiency test

Musculoskeletal:
ROM
hip
flex. 90/120
ext. 15
abd. 45
add. 30
int rot 40
ext rot 45
knee
flex. 130
ext. 0/15
ankle
plantar flex 45
dorsiflex 20
inversion 30
eversion 20
leg length

Neurological:
  Sensation
    L1
    L2
    L3
    L4
    L5
    S1
  Muscle strength
    hip flexion
    knee extension
    ankle dorsiflexion
    plantar flexion
  Tendon reflexes
    patellar
    Achilles
  plantar reflex
Rectal examination:
  Inspection
    perineum & perianal area
  Palpation
    sphincter tone
    tenderness
  injury:
    nodules
    prostate
    rectal wall

Mental status
  Appearance and behaviour:
    level of consciousness
    posture and motor behaviour
    dress, grooming, personal hygiene
    facial expression
    affect
  Speech and language:
    quantity
    rate
    volume
    fluency
    aphasia (pre)
  Mood
  Thought processes (logical, relevant, organized)
  Memory and attention:
    orientation (time, place, person)
    remote memory
    recent memory
    new learning ability
  Higher cognitive functions:
    information and vocabulary (general & specialised knowledge)
    abstract thinking.

APPENDIX 3

TECHNIKON NATAL

CHIROPRACTIC DAY CLINIC

REGIONAL EXAMINATION ----- THE KNEE

PATIENT : _____ AGE : _____

DATE : _____ FILE NO. : _____

X-RAY NO. : _____ PATH.LAB.NO. : _____

INTERN : _____ SIGNATURE : _____

CLINICIAN : _____ SIGNATURE : _____

HISTORY

MAIN COMPLAINT : _____

_____

_____

FUNCTIONAL IMPAIRMENT : _____

_____

_____

_____

MECHANICS OF INJURY : _____

_____

_____

_____

SITE OF PAIN : _____

_____

NATURE OF PAIN :

DURATION :

PREVIOUS INJURIES :

PREVIOUS TREATMENT :

ALLEVIATION OF PAIN :

EXACERBATION OF PAIN :

GAIT :

SWELLING :

SKIN :

RECREATIONAL/OCCUPATIONAL ACTIVITIES :

ADDITIONAL HISTORY INFORMATION :

OBSERVATION ------- STANDING AND SEATED

ANTERIOR VIEW

GENU VARUM : _____

GENU VALGUM : _____

SWELLING/BONY ENLARGEMENTS : _____

_____

PATELLAR POSITION : _____

_____

TIBIAL TORSION : _____

SKIN : _____

_____

LATERAL

GENU RECURVATUM : _____

PATELLA ALTA : _____

PATELLA BAJA : _____

SKIN : _____

_____

POSTERIOR VIEW

SWELLING : _____

_____

SKIN : _____

_____

GENERAL COMMENTS : _____

_____

_____

ACTIVE MOVEMENTS

FLEXION (0-135) _____

EXTENSION (0-15) _____

MEDIAL ROTATION (20-30) _____

LATERAL ROTATION (30-40) _____

PASSIVE MOVEMENTS --- NOTE END FEEL AND RANGE OF MOTION

FLEXION _____

EXTENSION _____

MEDIAL ROTATION _____

LATERAL ROTATION _____

PATELLAR MOVEMENT _____

_____

RESISTED ISOMETRIC MOVEMENTS --- THE KNEE

FLEXION _____

EXTENSION _____

RESISTED ISOMETRIC MOVEMENTS --- THE ANKLE

PLANTAR FLEXION _____

DORSIFLEXION _____

NOTE SPECIFIC MUSCLE INVOLVEMENT IF APPLICABLE

_____

_____

_____

GENERAL COMMENTS REGARDING MOVEMENT ASSESSMENT : _____

_____

_____

LIGAMENTOUS ASSESMENT

ONE-PLANE MEDIAL INSTABILITY

VALGUS STRESS (ABDUCTION)

- EXTENDED _____

- RESTING POSITION _____

ONE-PLANE LATERAL INSTABILITY

VARUS STRESS (ADDUCTION)

- EXTENDED _____

- RESTING POSITION _____

ONE-PLANE ANTERIOR INSTABILITY

LACHMAN TEST _____

ANTERIOR DRAWER TEST _____

ONE-PLANE POSTERIOR INSTABILITY

POSTERIOR "SAG" SIGN _____

POSTERIOR DRAWER TEST _____

ANTERIOMEDIAL ROTATORY INSTABILITY

SLOCUM TEST (FOOT LATERALLY ROTATED 15*)_____

ANTERIOLATERAL ROTATORY INSTABILITY

SLOCUM TEST (FOOT MEDIALLY ROTATED 30*)_____

MACINTOSH TEST _____

POSTEROMEDIAL ROTATORY INSTABILITY

HUGHSTON'S DRAWER SIGN _____

POSTEROLATERAL ROTATORY INSTABILITY

JACOB TEST _____

HUGHSTON'S DRAWER SIGN _____

TESTS FOR MENISCUS INJURY

MCMURRAY TEST _____

"BOUNCE HOME" TEST _____

O'DONOGHUE'S TEST _____

PLICA TESTS

MEDIOPATELLAR PLICA TEST _____

PLICA "STUTTER" TEST _____

HUGHSTON'S PLICA TEST _____

TESTS FOR SWELLING

BRUSH/STROKE TEST _____

PATELLAR TAP TEST _____

TESTS FOR CHONDROMALACIA PATELLA

CLARKE'S SIGN _____

OTHER TESTS

WILSON'S TEST (OSTEOCHONDRITIS DESSICANS)_____

FAIRBANK'S TEST (PATELLA DISLOCATION)_____

NOBLE COMPRESSION TEST (ITB FRICTION)_____

QUADRICEPS CONTUSION TEST_____

LEG LENGTH DISCREPENCY_____

_____

_____

_____

JOINT PLAY MOVEMENTS

BACKWARD MOVEMENT OF THE TIBIA ON THE FEMUR _____

FORWARD MOVEMENT OF THE TIBIA ON THE FEMUR _____

MEDIAL TRANSLATION OF THE TIBIA ON THE FEMUR _____

LATERAL TRANSLATION OF THE TIBIA ON THE FEMUR _____

DEPRESSION (DISTAL MOVEMENT) OF THE PATELLA _____

ANTERIO-POSTERIOR MOVEMENT OF THE TIBIOFIBULAR JOINT _____

PALPATION

ABNORMAL TENDERNESS _____

_____

SWELLING _____

_____

NODULES _____

_____

ABNORMAL TEMPERATURE _____

_____

REFLEXES AND CUTANEOUS DISTRIBUTION

PATELLAR REFLEX (L3,L4) _____

MEDIAL HAMSTRING REFLEX (L5,S1)_____

DERMATOMES

L2_____
L3_____
L4_____
L5_____
S1_____
S2_____
S3_____

RADIOGRAPHIC EXAMINATION

DIAGNOSIS :

TREATMENT PROTOCOL :

CLINICIAN'S COMMENTS :

APPENDIX 4

TECHNIKON NATAL
CHIROPRACTIC DAY CLINIC

REGIONAL EXAMINATION ----- THE HIP

PATIENT : _____ AGE : _____

DATE : _____ FILE NO. : _____

X-RAY NO. : _____ PATH.LAB.NO. : _____

INTERN : _____ SIGNATURE : _____

CLINICIAN : _____ SIGNATURE : _____

HISTORY

MAIN COMPLAINT : _____

_____

_____

FUNCTIONAL IMPAIRMENT : _____

_____

_____

MECHANICS OF INJURY : _____

_____

_____

DURATION : _____

_____

SITE OF PAIN : _____

_____

NATURE OF PAIN : _____

_____

ALLEVIATION OF PAIN :_____

_____

EXACERBATION OF PAIN :_____

_____

IS CONDITION STATIC, WORSENING, IMPROVING ?_____

WEAK OR ABNORMAL MOVEMENTS (SUBJECTIVE) :_____

_____

PREVIOUS INJURY :_____

_____

PREVIOUS TREATMENT :_____

_____

ADDITIONAL HISTORY INFORMATION :_____

_____

_____

_____

_____

_____

OBSERVATION

GAIT :_____

_____

_____

POSTURE :_____

_____

_____

WEIGHT BEARING :_____

_____

USE OF SUPPORT :_____

BALANCE : _____

_____

PROPRIOCEPTION (STORK STANDING TEST) _____

_____

SKIN : _____

_____

ANTERIOR VIEW:

BONY CONTOURS _____

SOFT-TISSUE CONTOURS _____

SWELLING _____

LATERAL VIEW

BUTTOCK CONTOUR _____

HIP FLEXION DEFORMITY _____

LUMBAR SPINE LORDOSIS _____

POSTERIOR VIEW

LUMBAR SPINE SCOLIOSIS _____

BONY CONTOURS _____

SOFT-TISSUE CONTOURS _____

EXAMINATION

ACTIVE MOVEMENTS

FLEXION (110-120) _____

EXTENSION (10-15) _____

ABDUCTION (30-50) _____

ADDUCTION (30) _____

LATERAL ROTATION (40-60) _____

MEDIAL ROTATION (30-40) _____

COMMENTS : _____

_____

_____

PASSIVE MOVEMENTS (NOTE END FEEL AND RANGE OF MOTION)

FLEXION _____

EXTENSION _____

ABDUCTION _____

ADDUCTION _____

LATERAL ROTATION _____

MEDIAL ROTATION _____

COMMENTS : _____

_____

_____

RESISTED ISOMETRIC MOVEMENTS

HIP FLEXION _____

HIP EXTENSION _____

HIP ABDUCTION _____

HIP ADDUCTION _____

HIP MEDIAL ROTATION _____

HIP LATERAL ROTATION _____

KNEE FLEXION _____

KNEE EXTENSION _____

COMMENTS _____

_____

NOTE SPECIFIC MUSCLE INVOLVEMENT IF APPLICABLE _____

_____

_____

JOINT PLAY MOVEMENTS

CAUDAL GLIDE (LONG LEG TRACTION) _____

COMPRESSION _____

LATERAL DISTRACTION _____

COMMENTS _____

_____

_____

SPECIAL TESTS

PATRICK-FABER TEST (hip pathology)_____

_____

TRENDELENBERG'S TEST (assess hip stability)_____

_____

CRAIG'S TEST (femoral anteversion)_____

_____

LEG LENGTH ASSESSMENT_____

_____

SIGN OF THE BUTTOCK (assess site of lesion)_____

_____

THOMAS TEST (hip flexion contracture)_____

_____

ELY'S TEST (rectus femoris hypertonicity) _____

_____

OBER'S TEST (ITB contracture) _____

_____

NOBLE COMPRESSION TEST (ITB friction syndrome) _____

_____

PIRIFORMIS TEST _____

_____

HAMSTRING CONTRACTURE TEST _____

_____

DERMATOMES

L1 _____

L2 _____

L3 _____

S3 _____

S4 _____

PALPATION

ANTERIOR ASPECT

ILIAC CREST _____

GREATER TROCHANTER _____

ASIS _____

INGUINAL LIGAMENT _____

FEMORAL TRIANGLE _____

HIP JOINT _____

SYMPHYSIS PUBIS _____

POSTERIOR ASPECT

ILIAC CREST _____

PSIS _____

ISCHIAL TUBEROSISTY _____

GREATER TROCHANTER _____

SACROILIAC JOINTS _____

LUMBOSACRAL JOINTS _____

SACROCOCCYGEAL JOINTS _____

COMMENTS :

RADIOGRAPHIC EXAMINATION

DIAGNOSIS :

TREATMENT PROTOCOL :

CLINICIAN'S COMMENTS :

APPENDIX 5

NUMERICAL 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 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

_____

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 .

_____

Appendix 6

Short-form McGill Pain Questionnaire (SF-MPQ)

RONALD MELZACK

Patients Name ..... Date .....

	NONE	MILD	MODERATE	SEVERE
THROBBING	0)	1)	2)	3)
SHOOTING	0)	1)	2)	3)
STABBING	0)	1)	2)	3)
SHARP	0)	1)	2)	3)
CRAMPING	0)	1)	2)	3)
GNAWING	0)	1)	2)	3)
HOT-BURNING	0)	1)	2)	3)
ACHING	0)	1)	2)	3)
HEAVY	0)	1)	2)	3)
TENDER	0)	1)	2)	3)
SPLITTING	0)	1)	2)	3)
TIRING-EXHAUSTING	0)	1)	2)	3)
SICKENING	0)	1)	2)	3)
FEARFUL	0)	1)	2)	3)
PUNISHING-CRUEL	0)	1)	2)	3)

In the above chart, please mark with a tick the nature of the pain which you feel best represents the pain(s) or sensation(s) you are experiencing. Please tick one column per line according to the severity of the pain, i.e. choose between (a) non, (1) mild, (2) moderate, (3), severe. You may tick more than one line, e.g: if pain is stabbing and sharp, tick both.

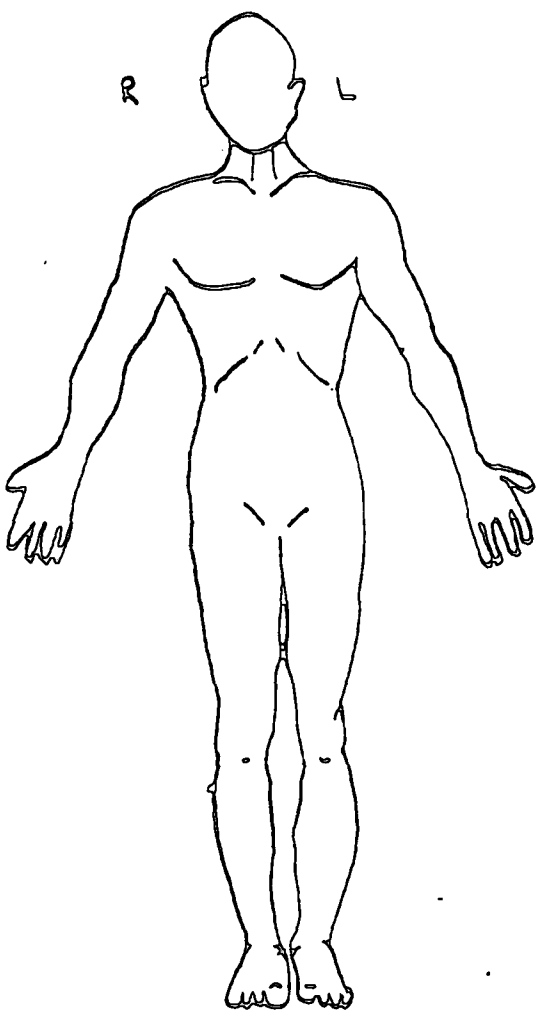
APPENDIX 7

PATIENT NAME: _____ FILE #: _____ DATE: _____

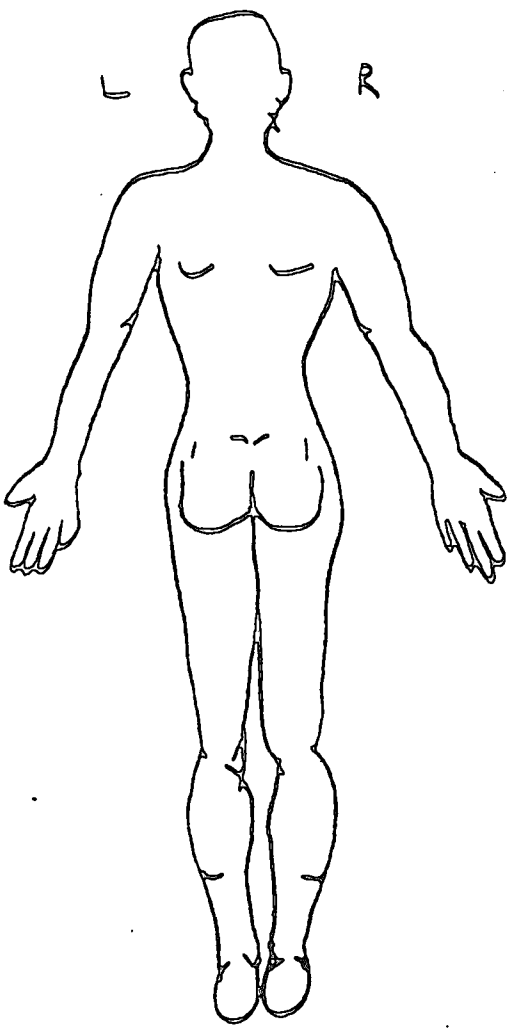
In the diagram provided below, please mark the areas on your body which you feel best represent the pain(s) or sensation(s) you are experiencing. Please include all areas. Use the symbols provided below.

SYMBOLS

numbness	=== ===	pins and needles	....
burning	XXX XXX	stabbing and sharp	//// ////
dull and aching	-++ -++	stiff and tight	ZZZ ZZZ



FRONT



BACK

P.T.O.

1.1 Pain Intensity

[ ] I have no pain
[ ] The pain is very mild at the moment
[ ] The pain is moderate at the moment
[ ] The pain is fairly severe at the moment
[ ] The pain is very severe at the moment
[ ] The pain is worst imaginable at the moment

1.2 Running

[ ] I have no pain during or after running
[ ] Pain comes on after running but does not restrict distance of speed
[ ] Pain comes during running but does not restrict distance or speed
[ ] Pain comes on during running and restricts distance and speed
[ ] Pain is so severe it prevents running
[ ] Pain is continuous during activities of daily living

1.3 Personal care (washing, dressing etc)

[ ] I can look after myself without causing extra pain
[ ] I can look after myself normally but is causes extra pain
[ ] It is painful to look after myself and I am slow and careful
[ ] I need some help but manage most of my personal care
[ ] I need help every day in most aspects of care
[ ] I do not get dressed, I wash with difficulty and stay in bed

1.4 Lifting

[ ] I can lift heavy weights without extra pain
[ ] I can lift heavy weights but it causes extra pain
[ ] Pain prevents me from lifting heavy weights off the floor, but I can manage if they are conveniently positioned, for example, on a table
[ ] Pain prevents me from lifting heavy weights, but I manage light to medium weights if they are conveniently positioned
[ ] I can lift very-light-weights
[ ] I cannot lift or carry anything at all

1.5 Walking

[ ] Pain does not prevent me from walking any distance
[ ] Pain prevents me from walking more than 2 kilometres
[ ] Pain prevents me from walking more than 1 kilometre
[ ] Pain prevents me from walking more than 0.5 kilometres
[ ] I can only walk using a stick or crutches
[ ] I am in bed most of the time and have to crawl to the toilet

1.6 Concentration

[ ] I can concentrate fully when I want to without difficulty
[ ] I can concentrate fully when I want to with slight difficulty
[ ] I have a fair degree of difficulty in concentrating when I want to
[ ] I have a lot of difficulty in concentrating when I want to
[ ] I have a great deal of difficulty in concentrating when I want to
[ ] I cannot concentrate at all

1.7 Work

[ ] I do as much work as I want to
[ ] I can only do my usual work, but no more
[ ] I can do most of my usual work, but no more
[ ] I cannot do my usual work
[ ] I can hardly do any work at all
[ ] I cannot do any work at all

1.8 Standing

[ ] I can stand as long as I want with no extra pain
[ ] I can stand as long a I want, but it give me extra pain
[ ] Pain prevents me from standing for more than 1 hour
[ ] Pain prevents me from standing for more than 30 minutes
[ ] Pain prevents me from standing for more than 10 minutes
[ ] Pain prevents me from standing at all

1.9 Social Life

[ ] My social life is normal and gives me no extra pain
[ ] My social life is normal but increases the degree of pain
[ ] Pain has no significant effect on my social life apart from limiting my more energetic interest, for example, dancing
[ ] Pain has restricted my social life to my home
[ ] I have no social life due to pain

1.10 Sleeping

[ ] I have no trouble sleeping
[ ] My sleep is slightly disturbed (less than 2 hours sleepless)
[ ] My sleep is mildly disturbed (1 - 2 hours sleepless)
[ ] My sleep is moderately disturbed (2 - 3 hours sleepless)
[ ] My sleep is greatly disturbed (3 - 5 hours sleepless)
[ ] My sleep is completely disturbed (5 - 7 hours sleepless)

1.11 Travelling

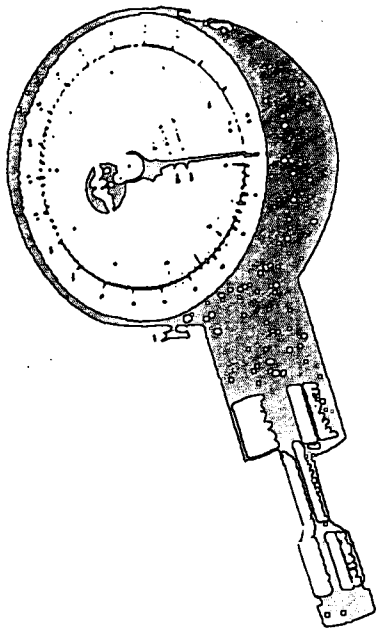
[ ] I can travel anywhere without extra pain
[ ] I can travel anywhere, but it gives me extra pain
[ ] Pain is bad but I manage trips over 2 hours
[ ] Pain restricts me to trips of less than 1 hours
[ ] Pain restricts me to trips under 30 minutes
[ ] Pain prevents me from travelling, except to the doctor or hospital

1.12 Recreation

[ ] I am able to engage in all my recreation activities with no pain at all
[ ] I am able to engage in all my recreation activities with some pain
[ ] I am able to engage in most, not all of my usual recreational activities due to pain
[ ] I am able to engage in few of my recreation activities due to pain
[ ] I cannot do any recreation activities at all

ALGOMETER READINGS

NO.	BEFORE TREATMENT	AFTER TREATMENT (kg/cm)
1		
2		
3		
4		
5		
6		
7		



Activator Methods, INC.
3714 E. INDIAN SCHOOL RD. PHOENIX, AZ 85018
(602) 224-0220

GENERAL INFORMATION

The algometer is most accurate in the range which is 75% from full scale. In the range below 25% of full scale, the gauge will give consistent readings, however, with less accurate results. This inaccuracy is inherent to the design of mechanical gauges. (Note: several studies have demonstrated reliability in a clinical setting.)

The algometer requires no lubrication or other form of service.

The face of the meter has no zero setting because the zero has no significance in the calibration or accuracy of the gauge.

CALIBRATION

Activator Methods certifies that all algometers have been properly calibrated and are accurate to ± 1% of full scale. The calibration of the algometer may be checked by attaching the pull hook and suspending test weights at ¼, ½, ¾, and full capacity in the vertical position. The weight of the plunger, flat tip, and pull hook (15 g.) should be subtracted from the test results. If it is determined that calibration is required, the instrument should be returned to the factory.

ALGOMETER INSTRUCTIONS

Background:

Pressure pain threshold (PPT) has been used by many authors to quantify palpatory pain findings for myofascial trigger points and pain over bone using an algometer (1-7).

Description:

The pressure algometer consists of a force dial which reads in pounds or kilograms and a 1 cm diameter rubber tipped stylus. Pain threshold is determined by the amount of force/cm² required for a person to first perceive pain.

Procedure:

Prior to recording the pain threshold, discuss the procedure with the patient. Before taking a measurement, you may wish to demonstrate the process to the patient by pressing the algometer into the palm of their hand.

1. Localize any sensitive areas you wish to measure by gentle but firm palpation.
2. Hold the meter in the palm of your hand between your thumb and index finger.
3. Place the rubber tipped stylus over the pre-determined trigger point or area of palpable tenderness you wish to measure. Make sure the force dial is perpendicular to the skin surface. Stabilize any nodular muscular regions between the middle and index finger of the indifferent hand.
4. Apply steady, gentle pressure at a rate of 1kg/cm²/sec. until the patient first feels pain and responds by saying "now."
5. Remove the stylus and record the value and locations of the tender areas in your notes or on a diagram for follow-up examination.
6. Reset the meter prior to making another reading.

References:

1. Fischer AA. Pressure algometry over normal muscles: Standard values, validity, reproducibility of pressure threshold. Pain 1986; 30:115-126.
2. Hsieh J, Hong CZ. Effect of chiropractic manipulation on the pain threshold of myofascial trigger point: a pilot study. Presented at the FCER's 2nd Annual International Conference on Spinal Manipulation, May 11-12, 1990, Washington, D.C.
3. Maloney P, Tepe R, Buerger D, et al. Evaluating the algometer as a diagnostic instrument. Presented at the FCER's 1st Annual International Conference on Spinal Manipulation, March 31-April 1, 1989, Washington, D.C.
4. Osterbauer PJ, Fuhr AW, DeBoer KF, et al. Preliminary clinical and biomechanical assessment patients with chronic sacroiliac syndrome. Presented at the FCER's 2nd Annual International Conference on Spinal Manipulation, May 11-12, 1990, Washington, D.C.
5. Reeves JL, Jaeger B, Graff-Radford SB. Reliability of the pressure algometer as a measure of myofascial trigger point sensitivity. Pain 1986; 24:313-21.
6. Vernon HT. Pressure pain threshold evaluation of the effect of spinal manipulation on chronic neck pain: a single case study. J Can Chiropr Assoc 1988; 32(4):191-4.
7. Vernon HT, Acker P, Burns S., et al. Pressure pain threshold evaluation of the effect of spinal manipulation in the treatment of chronic neck pain: a pilot study. J. Manipulative & Physiol Ther 1990; 13(1):13-16.

THIS INSTRUMENT CARRIES A ONE YEAR WARRANTY FROM DATE OF PURCHASE.

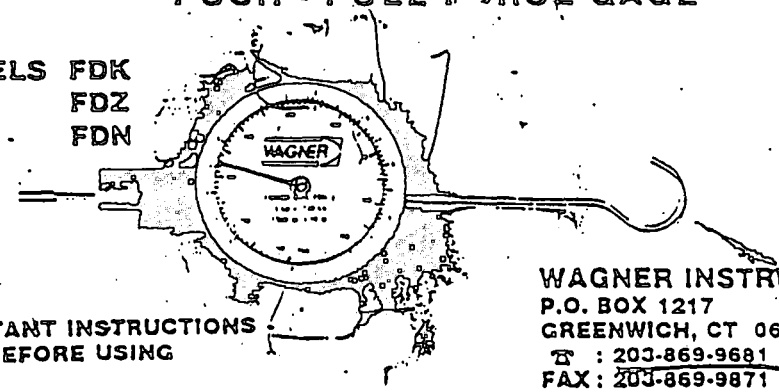
FORCE DIAL
CERTIFICATE OF CALIBRATION

WAGNER INSTRUMENTS certifies that all FORCE DIALS are calibrated at the factory to meet the specified accuracy of ± 1% of full scale, advertised in our current catalog.

QUALITY CONTROL DIRECTOR

FORCE DIAL™
PUSH - PULL FORCE GAGE

MODELS FDK
FDZ
FDN



IMPORTANT INSTRUCTIONS
READ BEFORE USING

WAGNER INSTRUMENTS
P.O. BOX 1217
GREENWICH, CT 06836 U.S.A.
☎ : 203-869-9681
FAX: 203-869-9871

Complete list of available FORCE DIALS

FDK

DECIMAL POUND / GRAM GRADUATIONS

Model	Capacity/Graduations					
FDK 025	.25 LB x	.002 LB/	100 G x	1 G		
FDK 050	.50 LB x	.005 LB/	200 G x	2 G		
FDK 1	1 LB x	.010 LB/	500 G x	5 G		
FDK 2	2 LB x	.020 LB/	1000 G x	10 G		
FDK 5	5 LB x	.050 LB/	2500 G x	25 G		
FDK 10	10 LB x	.100 LB/	5 KG x	50 G		
FDK 20	20 LB x	.250 LB/	10 KG x	100 G		
FDK 40	40 LB x	.500 LB/	20 KG x	200 G		
FDK 60	60 LB x	.500 LB/	28 KG x	250 G		

OUNCE / GRAM GRADUATIONS

Model	Capacity/Graduation			
FDK 4	4 OZ x	1/32 OZ/	100 G x	1 G
FDK 8	8 OZ x	1/16 OZ/	200 G x	2 G
FDK 16	16 OZ x	1/8 OZ/	500 G x	5 G
FDK 32	32 OZ x	1/4 OZ/	1000 G x	10 G
FDK 80	5 LB x	1 OZ/	2500 G x	25 G
FDK 160	10 LB x	2 OZ/	5 KG x	50 G

FDN

NEWTON / GRAM GRADUATIONS

Model	Capacity/Graduation			
FDN1	1N x	.01N/	100G x	1G
FDN2	2N x	.02N/	200G x	2G
FDN5	5N x	.05N/	500G x	5G
FDN10	10N x	.1N/	1000G x	10G
FDN20	20N x	.2N/	2000G x	20G
FDN50	50N x	.5N/	5KG x	50G

GENERAL

Your FORCE DIAL should not be used to measure forces below 25% of full scale since true accuracy is degraded as readings decrease from full scale. Before placing the FORCE DIAL into service it is also recommended to test for accuracy according to procedures found in the CALIBRATION section of this manual.

Model FDK FORCE DIALS have no zero on the dial, since setting the pointer at zero has no significance in calibration or accuracy: see CALIBRATION for details.

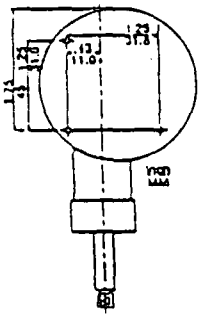
Lubrication of the FORCE DIAL is not recommended.

IMPORTANT

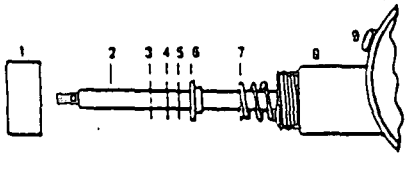
To prevent damage, keep an implement/accessory on the plunger even when the gage is not in use and when using the pull hook. This provides a positive stop and prevents the plunger from being pushed too far.

MOUNT

Your FORCE DIAL may be mounted with three #6 (.138 in/3.5 mm O.D.) sheetmetal screws using the hole pattern shown below. The three dimples on the rear housing will assist in starting the screws. Sturdy posts are located internally behind the dimples to accept the screws. The screws should penetrate no more than 3/8 inches or 10 mm.



PARTS



(1) Retainer	(5) Calibration Washers	(8) Case
(2) Plunger		(9) Push Button
(3) Disc	(6) Plate	(10) Crystal *
(4) Clip	(7) Spring	(11) Pointer *

ACCESSORIES: *

(12) Flat Tip (thru 2 LB / 1000 G / 10 N)
(13) Flat Tip (5 LB / 2500 G / 20 N & up)
(14) Long Rod (thru 2 LB / 1000 G / 10 N)
(15) Long Rod (5 LB / 2500 G / 20 N & up)
(16) Pull Hook (thru 2 LB / 1000 G / 10 N)
(17) Pull Hook (5 LB / 2500 G / 20 N & up)

CALIBRATION

The calibration of the FORCE DIAL may be checked by attaching the pull hook and suspending test weights at 1/4, 1/2, 3/4, and full capacity in the vertical position. The weight of the plunger, flat, tip and pull hook (.03 LB, 17/32 OZ, 15 G) should be subtracted from test results. If it is determined that recalibration is required the instrument should be returned to the factory.

IMPLEMENT WEIGHT ADJUSTMENT

The FORCE DIAL is calibrated for use in the horizontal position. When using low capacity models - thru 2 LB/ 1000 G/ 10 N - in the vertical position, add or deduct the weight of the implements used from your readings, as follows:

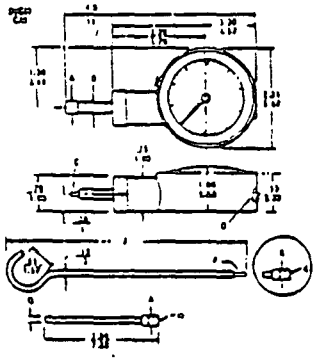
WEIGHT OF IMPLEMENTS:

- Plunger: .015 LB/ 1/4 OZ/ 7 G
- Flat Tip: .004 LB/ 1/16 OZ/ 2 G
- Long Rod: .009 LB/ 5/32 OZ/ 4 G
- Pull Hook: .013 LB/ 7/32 OZ/ 6 G

ADJUSTMENT:

USE	WITH	+/-
Pushing Down	Plunger/Flat Tip	+9 G
Pushing Down	Plunger/Long Rod	+11 G
Pulling Down	Plunger/Flat Tip/Hook	+15 G
Pushing Up	Plunger/Flat Tip	-9 G
Pushing Up	Plunger/Long Rod	-11 G
Pulling Up	Plunger/Flat Tip/Hook	-15 G

DIMENSIONS



High and low capacity models differ slightly in design. The lettered dimensions above, along with the corresponding measurements and comments shown below identify these small variations.

All dimensions are approximate.

Low Capacity (thru 2 LB / 1000 G)		High Capacity (5 LB / 2500 G & up)	
A .19"	.45 cm	A .26"	.65 cm
B .12"	.3 cm	B .24"	.6 cm
C M 3	male	C M 4	male
D M 3	male	D M 3	female
E M 3	female	F M 3	male
G .12"	.3 cm	G .14"	.35 cm
H M 3	female	H M 4	female
J 2.8"	7.1 cm	J 3.4"	8.6 cm

PATIENT CONSENT FORM

This is to confirm that I _____ am willing to participate in the research dissertation of THANDI SAUNDERS.

I undertake to the best of my ability to adhere to the designated program and to comply with the requests of the researcher.

I also understand that all personal information is strictly confidential.

_____ _____

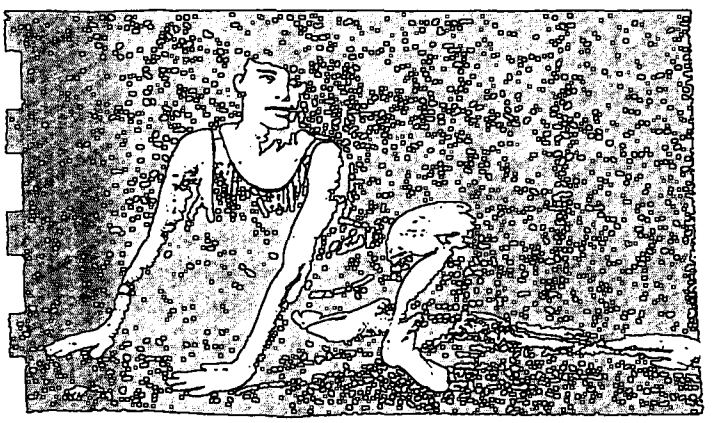
Date (Patient's signature)

_____

(Witness)

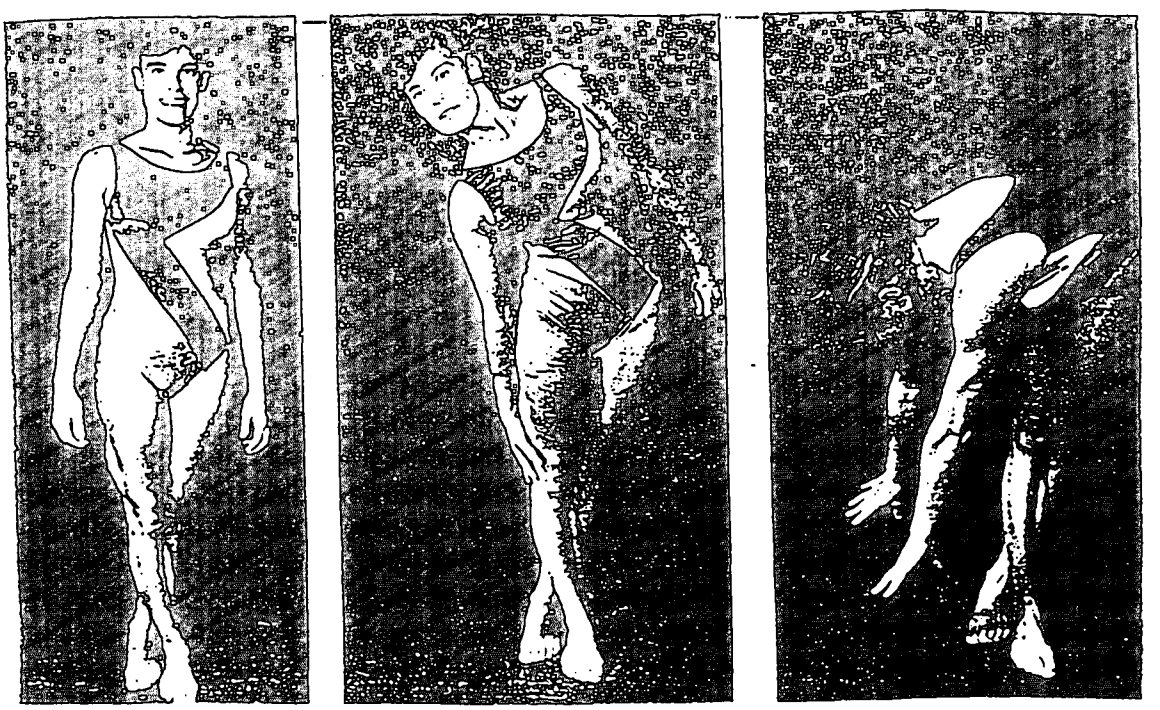
_____

(Witness)



Stretches for Iliotibial Band Friction Syndrome

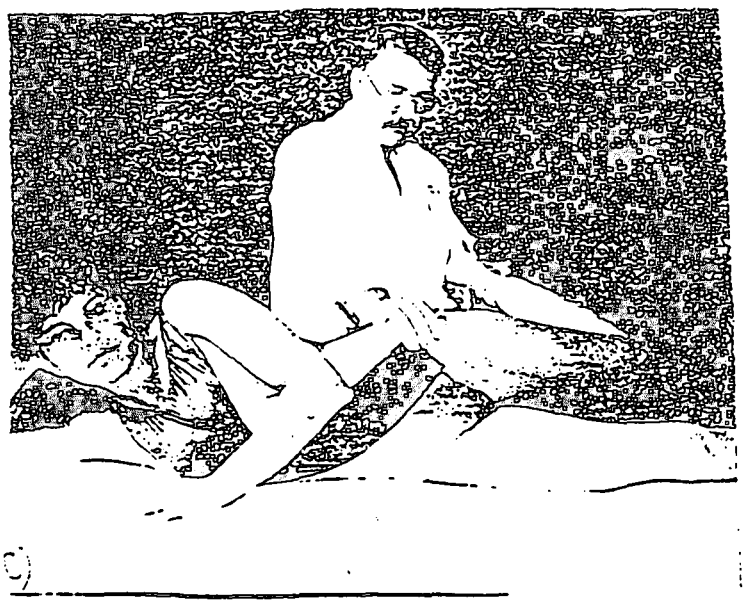
A) Athlete lies on affected side, hips and knees extended
b) Using the arms, the patient side-flexes
c) Opposite foot may be used to assist stabilization and maximize the stretch.



a) Both knees are in extension and the legs are crossed at the ankle.
b) With the knees locked, the athlete side flexes as much as possible to the unaffected side
c) Alternatively the patient may rotate the waist away from the affected side, flexing the trunk and attempting to touch the heel of the affected leg.

A)

B)



C)

D)

Appendix 12B

Ober's Test : The patient lies in the sidelying position with the affected side uppermost

a) The examiner grasps the ankle of the affected leg
b) The examiner then passively abducts and extends the patient's upper leg with the knee flexed to 90°
c) A normal test - the knee falls into adduction
d) A tight iliotibial band will cause the lower limb to remain abducted and not fall to the bed

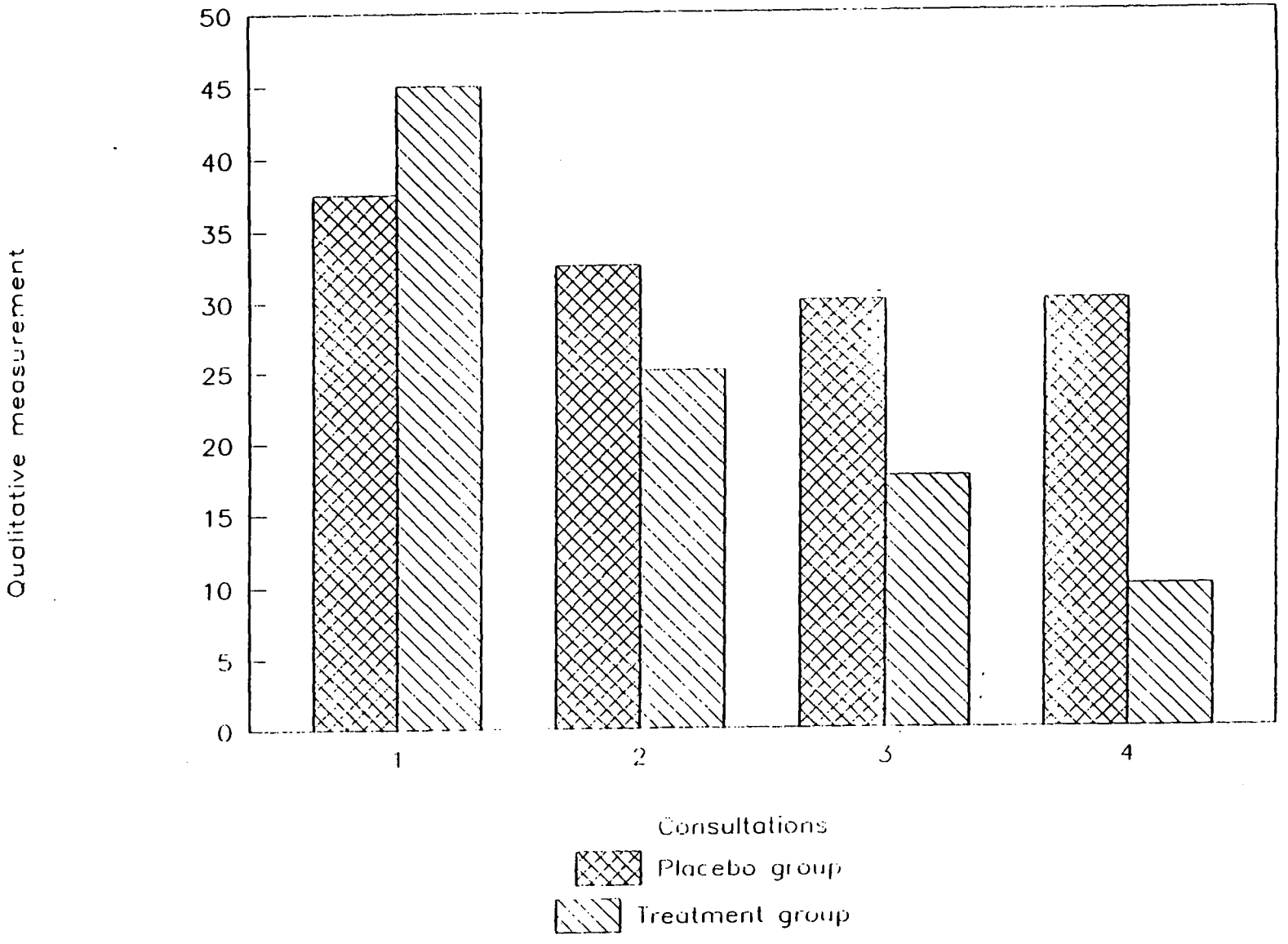
Appendix 13

SEVERITY OF OVERUSE SYNDROME (Reid, 1992: 424-429).

Grade I	-	Pain comes on after running but does not restrict distance or speed
Grade II	-	Pain comes on during a run but does not restrict distance or speed
Grade III	-	Pain comes on during a run and restricts distance or speed
Grade IV	-	Pain is so sever, it prevents running
Grade V	-	Pain is continuous during activities of daily living

Comparison with respect to the

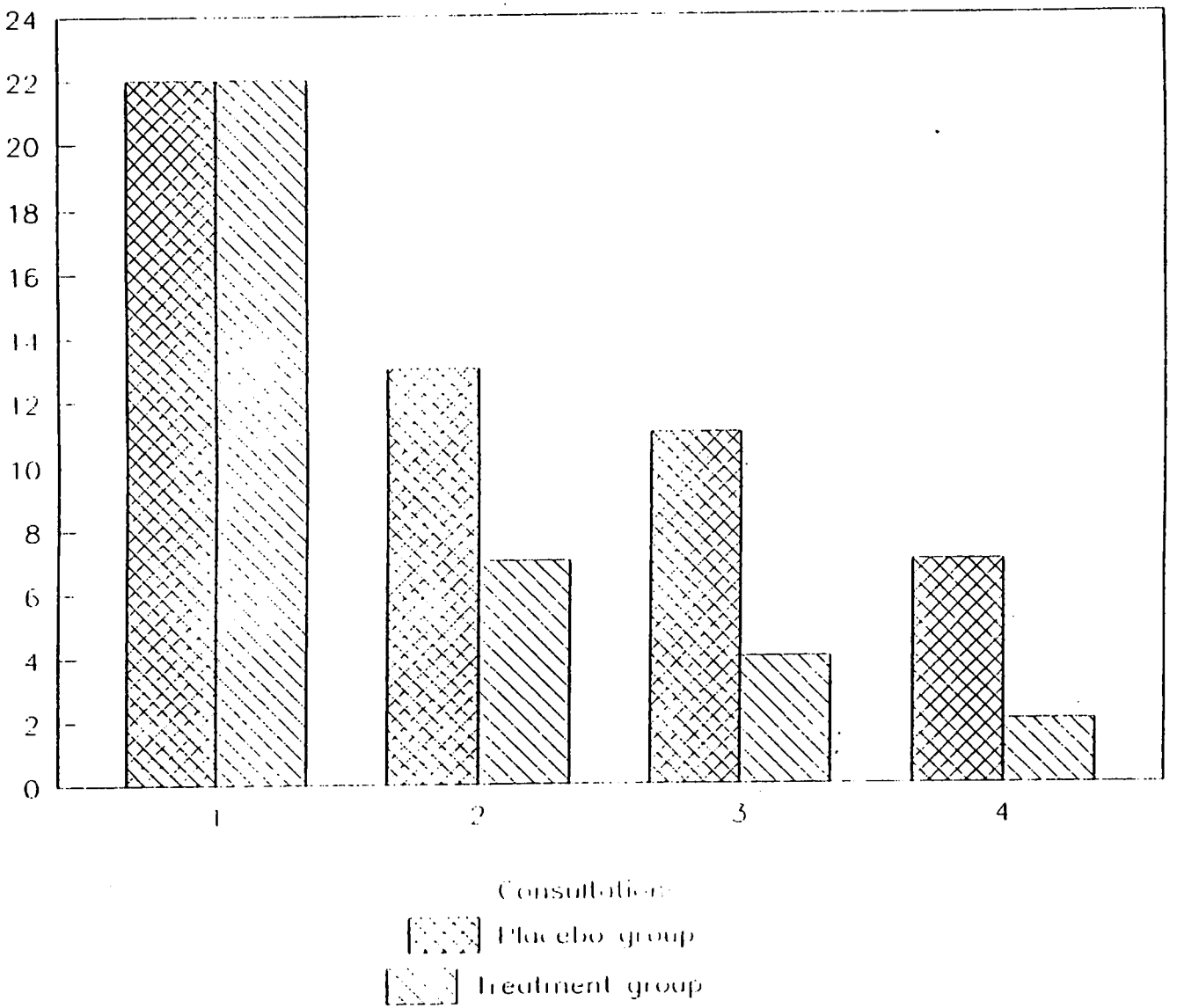
Numerical Pain Rating Scale 101



APPENDIX 14

Comparison with respect to McGill Pain

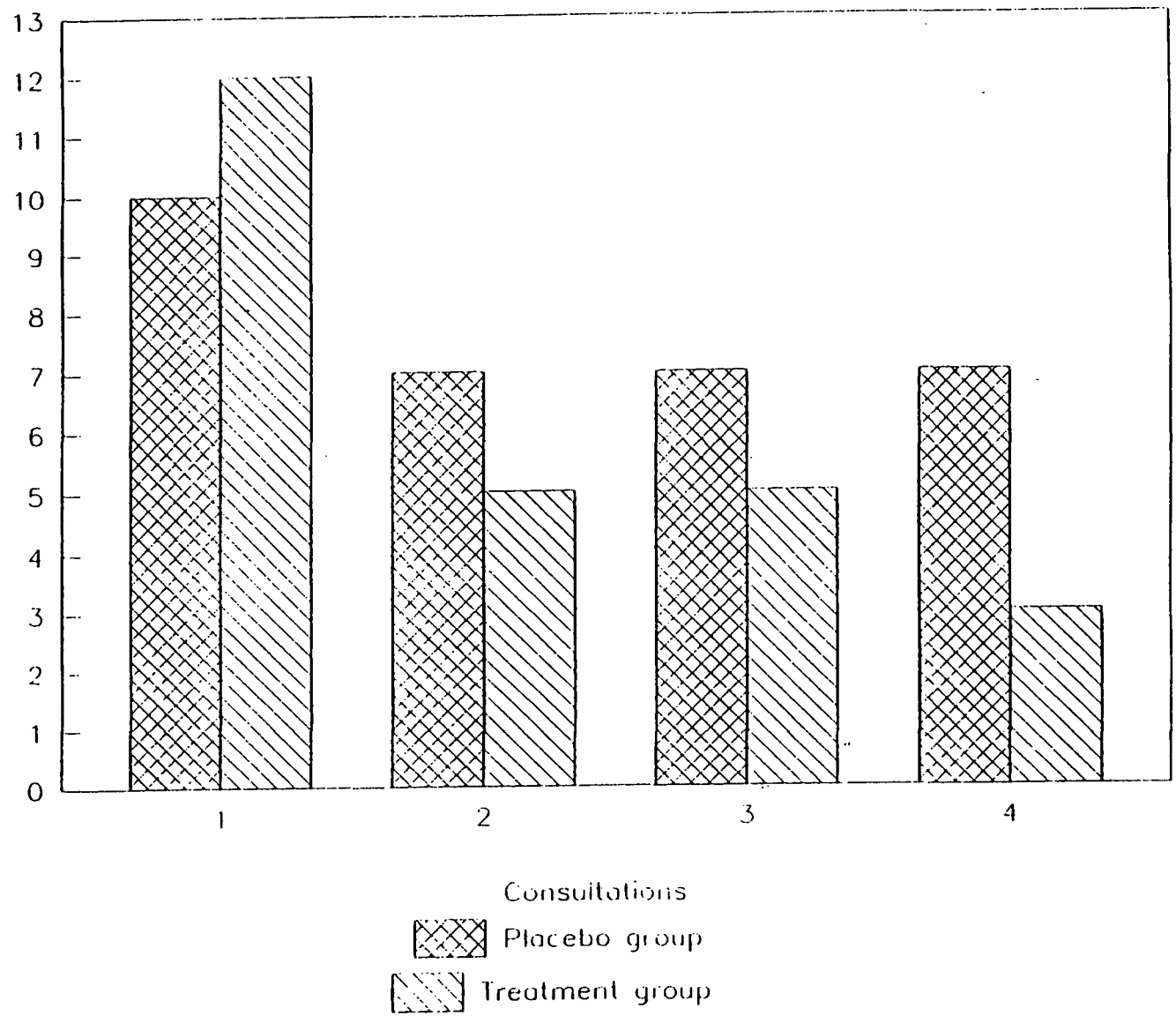
Questionnaire (median scores)



APPENDIX 15

Comparison with respect to

Pain Disability Index



APPENDIX 16

APPENDIX 17

Pressure in Kgs per square cms

ALCOMETER READINGS

Median values