QUADRICEPS INHIBITION POST ARTHROSCOPIC MENISECTOMY

THE INFLUENCE OF ARTHROSCOPIC MENISECTOMY AND POST SURGICAL TRANSCUTANEOUS ELECTRICAL NERVE STIMULATION ON QUADRICEPS STRENGTH AND MOTOR UNIT ACTIVATION

By

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ABSTRACT

Reflex inhibition of the quadriceps muscle group is a frequent and significant consequence of knee trauma, disease and surgical insult. The resultant quadriceps atrophy can be expected to delay rehabilitation and render the joint vulnerable to repeated injury resulting in capsular and synovial thickening, effusion and pain. A major purpose of this study was to examine the degree of quadriceps inhibition experienced by patients who undergo arthroscopic menisectomy. A secondary goal of this study was to investigate the efficacy of transcutaneous electrical nerve stimulation on the relief of reflex inhibition. Tests were performed on 12 patients prior to, and on day 1 and day 2 post surgery. True and placebo treatments of transcutaneous electrical nerve stimulation were administered on day 1 and day 2 post surgery. Measurements were made on the injured and normal limb with the knee fixed at 38° of flexion. Motor unit activation was determined by the twitch interpolation technique. Reduced motor unit activation was considered indicative of quadriceps reflex inhibition. Testing demonstrated that at all times the injured leg was weaker than the normal leg ($p=.001$).

Following surgery, strength of the injured limb was significantly less than its pre operative score ($p=.01$). No significant recovery of strength was observed during the first two days following surgery. Injured legs were characterized by significantly lower motor unit activation at all times of testing ($p=.003$). Following surgery, motor unit activation for the injured leg was significantly lower than its pre operative value ($p=.01$). By day 2 post surgery, motor unit activation had recovered ($p=.05$) and was similar to the pre operative values for that leg. Transcutaneous electrical nerve stimulation had no effect on strength or motor unit activation. Recovery following arthroscopic surgery is characterized by an initial loss of strength and motor unit activation. By day 2, isometric strength remains depressed, however motor unit activation returns to pre surgery levels.

FOREWORD

In 1986 the Graduate Council of McMaster University approved submission of thesis manuscripts in a form similar to that which would be submitted to a scientific journal for publication. This thesis represents the first within the AHB programme to follow this format.

This thesis is presented in 3 main sections: Chapter I represents a review of literature related to the phenomenon of reflex inhibition; Chapter II is the thesis research presented in manuscript form; Appendix I is a presentation, of a pilot study on the efficacy of TENS on the performance of an isometric strength test. A second appendix presents the results of a pilot study to determine the reproducibility of the test procedures used in data collection.

By approving this optional presentation format the AHB Graduate Committee hopes to encourage a writing style that will prepare for career related writing. It is hoped that this approach to thesis presentation will lead to a concise manuscript suitable for publication with few revisions.

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CHAPTER I

REFLEX INHIBITION

1.1 INTRODUCTION

The strength of a maximum voluntary contraction is partly dependent upon optimal neural activation of the involved muscles. This state of activity is in turn a function of full descending drive and reflexive events at the related spinal segment. In this manner, peripheral stimuli may facilitate or inhibit voluntary drive to a muscle group (25). "Reflex inhibition" is an event in which stimuli arising from a joint reflexively reduce motor unit activation of specific muscles surrounding that joint. Such inhibition is commonly observed following injury to the knee joint where it is characterized by inhibition of the quadriceps and facilitation of the hamstrings. The following review will, if not otherwise indicated, refer to

reflex inhibition as it occurs at the knee.

Reflex inhibition is a potent complicating factor in articular injury and disease processes. A group of patients with minor medial collateral ligament lesions exhibited altered EMG activity indicative of quadriceps inhibition for up to 30 days post injury (47). Following arthrotomy with menisectomy, quadriceps inhibition has been found to be 80 - 90 % of pre surgical values in the first 24 hours post surgery, with significant inhibition (35 - 40 %) persisting for at least two weeks following surgery (42). It is considered that such inhibition endured over a prolonged period will contribute to muscle atrophy and seriously retard the rehabilitation process.

Although reflex inhibition has been studied most frequently following knee injury and surgery, it has also been reported following elbow dislocation (8), and may be assumed to be present following injury to any peripheral joint. Its existence has significant implications in cases of rheumatoid arthritis or osteoarthitis. Management of the patient with these problems has typically involved the use of oral anti-inflammatory medications. An adjunctive treatment technique is the use of Transcutaneous Electrical

Nerve Stimulation (TENS). TENS is an electrotherapeutic modality used primarily for its analgesic effect. Some evidence exists that supports the efficacy of oral anti-inflammatories in reduction of reflex inhibition (35,36). It is not clear if TENS can effectively reduce the influence of this reflex.

Various investigators have attributed the source of reflex inhibition to pain (49), capsular compression (45), ligament stress (3,38,49) and joint effusion (12,46,48). Each of these investigators agrees that the critical stimuli arise from within the joint in question. Consequently, this review will begin with a description of the neuroreceptor system of synovial joints. Following this, the experimental evidence describing the existence and qualities of reflex inhibition will be reviewed and finally the possible role of Transcutaneous Electrical Nerve Stimulation, in relief of inhibition will be discussed.

1.2 Joint Innervation

It is generally agreed that the same trunks of those nerves whose branches supply the groups of muscles that move the knee also furnish a distribution to the interior of that joint (27,31,56). Among those authors reviewed, variations

in classification and details of articular innervation were found; however, it is agreed that those nerves listed in Table 1 contribute the major nerve supply to the knee joint. These articular supplies are augmented by numerous non-specific branches from the nerves to sartorious and quadriceps. A few smaller branches from the cutaneous nerves supplying the skin over the joint can also be found travelling into the knee (27,56).

TABLE 1a

NERVES CONTRIBUTING TO KNEE JOINT INNERVATION

Posterior Tibial Nerve
Posterior Division of the Obturator Nerve
Femoral Nerve
Common Peroneal Nerve
Saphenous Nerve

The histological and neurological qualities of the

nervous endorgans that exist in synovial joints are well described (9.11,16,45,56). As expected, these descriptions vary in terms of investigative technique and experimental model. Most investigators have tried to relate articular receptors to those nerve endings found in non-articular tissue. This approach invites preconceived bias when interpreting experimental findings. In 1967 Wyke (56) presented an orderly and well defined classification system which groups the various receptor types into arbitrary numerical catagories (Types I to IV). Each of the receptor types described by previous investigators are included in Wyke's model. This section will summarize the classification system proposed by Wyke and experimental work by other authors supporting Wyke's assertions will be noted, where appropriate. Review of these publications will reveal differences in technique and terminology; however, the histological and physiological properties of the receptors will be similar.

The various nerve fibers that together constitute the articular nerves have been subdivided, according to size, into 3 groups. Like the 4 types of joint receptors, Wyke has identified a specific functional correlate for Group I, II and III nerve fibers. These functions will be

discussed with reference to the end organs that are supplied by the nerve group.

1.3 Type I Receptors Ovoid shaped receptor cells have been located in the superficial layers of the fibrous capsule of the knee (21,22,23). These static/dynamic mechanoreceptors are arranged in tri-dimensional clusters on those aspects of the joint that undergo greatest changes in stress during natural joint movement. Skoglund describes a very similar end organ which he calls a Ruffini nerve ending (45). Type I receptors are low threshold and slow to adapt once activated (22,23,45). Wyke (1966) notes that these fibers respond to mechanical stresses applied to the area of the joint in which they lie (56).

As would be expected Type I receptors respond vigorously to active or passive joint movement. When mechanical stresses act upon the joint capsule, these receptors respond with an abrupt rise or fall in their discharge rate. The intensity of the discharge activity is dependent upon the degree of capsular stress and the particular area of the capsule involved (23).

Since Type I receptors have low thresholds and are

slow to adapt, these cells provide a low level of constant activity. An immobile joint will present with a Type I discharge rate of 10 - 20 pulses per second (24,45,56). As well as being sensitive to the relatively gross stresses of joint movement these cells may be provoked by subtle mechanical changes occurring within the joint. Skoglund has found that once these cells are activated they may continue to fire for 4 to 5 hours (45). Fluctuating tone in periarticular musculature or changes in the intra-articular to atmospheric pressure gradient is enough to cause a response in the Type I cells (45,56). These findings are based on studies of the action potentials observed in exposed articular nerves in cats as the knee is moved or otherwise stimulated.

Type I receptors are considered to be static and dynamic mechanoreceptors. These cells signal joint movement, the direction of the movement, its amplitude and velocity. They are supplied by Group II, myelinated, parent axons (23,45,56). These nerves are 6-9 μ in diameter and enter the joint capsule as a member of the articular nerves. Wyke defines Group II fibers as those between 6 and 12 μ in diameter. All such fibers are mechanoreceptor afferents. Group II fibers constitute 45 - 50 % of the total afferent

fibers in a articular nerve.

1.4 Type II Receptors Type II receptor cells are elongated, conical corpuscles. These receptors may be found near Type I cells as well as in the deeper layers of the capsule. Type II's often lie in close proximity to the articular blood vessels and within articular fat pads (22,23,56). Skoglund has described these endorgans as being modified Vater-Pacini corpuscles (45). These cells have been characterized as low threshold, rapidly adapting, mechanoreceptors. As such, Type II receptors are well suited to signal joint acceleration and deceleration. Unlike Type I receptors they are inactive in resting joints (23,45,56).

Type II receptor cells are supported by Group II, myelinated, afferents (23,56). Typically, the afferent that supplies a cluster of Type II cells is between 9 and 12 μ in diameter. These parent nerve cells fall within the same Group classification as those innervating Type I receptors; however, they represent the largest of those neurons in this catagory. This quality implies that Type II receptors are supported by afferents with a greater conduction velocity than those that run to Type I receptors.

1.5 Type III Receptors Type III receptors are described as the articular homologue of the Golgi Tendon Organ. Histological studies have determined that these cells are confined to the joint ligaments (22,23,45,56). Type III cells are high threshold cells that slowly adapt once activated. These cells are silent in the resting joint and only become active if the ligament is subjected to considerable mechanical stress. Upon Type III discharge, an action potential is propagated along a large diameter (13 - 17 μ), Group I afferent. These afferents represent less than 10% of the total composition of an articular nerve (23,56).

1.6 Type IV Receptors Type IV joint receptors are found throughout the fibrous capsule and adjacent periosteum. These cells have also been identified in articular fat pads and in the adventitial sheaths of the articular blood vessels. Type IV receptors are free nerve endings that form lattice like plexuses within the joints (22,23,45,56). They are absent in synovial tissue and joint menisci (22,23,56). Type IV cells constitute the endorgans of the articular pain pathways. They are sensitive to

marked mechanical deformation and/or chemical irritation (56).

These receptors are supported by myelinated and unmyelinated nerve fibers between 2 and 5 μ in diameter. Group IV end organs are supported by afferents that represent 45% of the total fibers in an articular nerve. This category of nerves have been labelled as group III by Wyke (23,56).

Group IV nerves are largely afferent in nature, however a small proportion of these nerves consist of visceral efferents. These unmyelinated fibers are of sympathetic origin and innervate the articular blood vessels (23,56). Wyke (56) cautions that no evidence exists to substantiate of the presence of secretomotor fibers within the articular nerves. He further states that there is no evidence of direct nervous influence on the production of synovial fluid. Wyke's work is based on post mortem anatomical studies of human and animal joints. These findings have since been challenged by Ferrell and Russell in 1985 (18). This group demonstrated, in a feline model, that stimulation of the nociceptor afferents results in a significant increase in the production of synovial fluid.

It is generally agreed that joint mechanoreceptors are major contributors to postural and kinaesthetic sensation (2,27,56,54). This afferent system is also capable of reflexively influencing periarticular muscle tone (7,9,11,20,21,22,23,45,56). Such influence may be facilitory or inhibitory and is generally reciprocally coordinated among the muscle groups acting about a joint.

Wyke states that the low threshold, quickly adapting, Type II mechanoreceptors not only signal joint movement but also serve as "boosters". Type II cells briefly facilitate the prime movers of a joint as movement commences while inhibiting antagonistic muscle groups. In this manner the muscle is assisted in overcoming the inertia of the immobile parts. Due to their rapid adaptation to local stimuli, Type II cells are not able to contribute to the perception of static joint posture (56).

Type II cell function is complemented by the slowly adapting Type I mechanoreceptors. Type I receptors exert a tonic influence over the reflex regulation of the tone of the periarticular muscles. These receptors play key roles in the establishment and maintenance of a condition of

reflex inhibition.

1.7 THE ROLE OF PAIN

Investigators have often considered articular pain as a necessary stimulus to provoke reflex inhibition, (28,42,47,49,57). This hypothesis implicates Type IV joint endorgans as the critical sensors in the afferent system. Wyke's (56) work, involving the temperomandibular joint, has determined that Type IV discharge provokes "intense non adapting motor unit responses simultaneously in all muscles related to the joint". In peripheral joints the pattern of response to a painful stimulus is one of flexor facilitation and extensor inhibition.

Clinical and laboratory studies have confirmed that joint pain is not a necessary condition for the establishment of reflex inhibition (8,12,26,42,46,51). Shakespeare and associates note that following knee surgery, inhibition was still severe even though pain was only mild or absent (42). Use of intra-articular anaesthesia effectively blocked the sensation of pain without reducing the experience of inhibition. These results are contrary to Stener's (47) report which indicated that noxious stimuli

may cause reflex inhibition. Reflex inhibition provoked through the experience of pain is of short duration. Such a response is likely to be equivalent to a flexor withdrawal response. This reaction is quite distinct from the chronic inhibition observed following joint injury and in the absence of pain.

Despite these findings, clinical management of reflex inhibition often includes medicine and modalities intended to relieve pain. Clinicians are cautioned that severe inhibition may exist even in the absence of joint pain.

1.8 JOINT COMPRESSION

Ekholm and colleagues (16) used a feline model to determine that inhibition of the knee extensors and facilitation of the flexors will result when the joint capsule is pinched. Stener (3,49) provoked a similar response by applying gentle mechanical stimulation to the exposed joint capsule.

According to Wyke, such stimuli would elicit activity from Type I and Type II joint mechanoreceptors. An

earlier study (45) determined that pressure applied to the joint capsule causes two distinct responses from the joint receptors. The first response is a low threshold, rapidly adapting discharge, provoked only during a change in the tension of the joint capsule. This response may be attributed to the Type II end organs described by Wyke. The second response is also low threshold; however, once activated a very long adaptation period follows. Skoglund notes that these receptors maintained a steady firing frequency for hours. Wyke's model would credit such a response to the Type I end organs.

Stimulation of Type I and II joint mechanoreceptors leads to inhibition of the knee extensors and facilitation of the knee flexors. The prolonged adaptation process exhibited by Type I end organs results in persistent inhibition despite favourable changes in the joint environment.

Capsular compression must be considered an infrequent event among a clinical population. Surgical trauma, capsular sutures, or articular staples may supply the necessary stimulus for reflex inhibition. Stress over a capsular band or adhesion may provide a form of capsular

compression. Reflex inhibition is known to be present in joints that have none of these conditions (46).

1.9 LIGAMENT STRESS

In 1959 Stener's group investigated the muscular response to an abnormal articular event (3,38,49). Their goal was to determine the existence of a ligamento-muscular protective reflex. It was contended that, besides acting as obstacles to abnormal movement, ligaments were also responsible for reflexively activating those muscles capable of opposing the abnormal movement. This reaction would be mediated via ligamentous tension receptors analogous to Wyke's Type III end organs. In extensive testing of normal knees, no muscular responses could be elicited despite strong valgus stresses intended to challenge the medial collateral ligament. Earlier work with a feline model confirmed that similar stresses were more than adequate to provoke discharge from the Type III receptors and that this discharge had no measurable influence on the periarticular musculature. Using the same animals, a vivid contrast in response was elicited with gentle mechanical stimulation of the joint capsule. Pressure applied through a glass rod caused an immediate inhibition of the extensors and

facilitation of the flexors. Capsular receptors exert a greater influence over the periarticular musculature.

Subsequently, Stener (47) tested EMG responses in human subjects with injured medial collateral ligaments. Adduction stresses applied to these joints produced vigorous responses in the knee flexors and inhibition of an extensor. An identical response could be triggered if pressure was applied to the area over the lesion. Four studies were made before and after administration of a local anaesthetic. In all cases reflexive muscular responses were absent following anaesthesia. Stener considers reflex inhibition to be a result of a nociceptive flexion reflex arising from the injured ligament. This interpretation is in conflict with studies which have established the dissociation of pain and inhibition.

Stener reported that the majority of patients included in his study were unable to fully extend their knees. This indicates that a significant degree of inhibition was present before testing began. It is possible that any inhibition provoked was due to alteration of joint position and mechanical stimulation of the joint capsule rather than a noxious event. Another interpretation might

be that use of a local anaesthetic not only blocked Type IV joint receptors but also Type I and II mechanoreceptors (42). Therefore, reflex inhibition might have been effectively abolished due to blockade of mechanical stimuli rather than to removal of noxious stimuli. The role of ligamentous stress in production of reflex inhibition thus remains controversial.

1.10 JOINT EFFUSION

It is a common clinical observation that patients with a tense joint effusion experience an inability to fully activate their quadriceps. The association of joint effusion and reflex inhibition has been confirmed in several studies (12,42,43,44,46,50,51).

Early investigation of this issue implied that a very large joint effusion was necessary to create a condition of quadriceps inhibition (12,29). Volumes of fluid as great as 300 ml were infused into knee joints to provoke reflex inhibition. Work by Spencer's group (46) indicates that these gross changes in the intra-articular environment are not necessary to note inhibition. Effusions as small as 20-30 ml produce significant inhibition of the

alpha motorneuron pool. Stokes and Young (50) note that, even in the absence of clinically detectable joint effusions, inhibition persists. In their studies aspiration of joint effusion always reduced inhibition but rarely abolished it. Based on this finding, Stokes and Young suggest that synovial inflammation and congestion is enough to trigger reflex inhibition. Spencer and co-workers found that reduction of effusion results in immediate but incomplete return of function. Further evidence from recent studies demonstrates that anti-inflammatory medication enhance recovery following knee surgery (35,36).

The relatively subtle influence of a 10 ml increment or decrement in knee effusions indicates that reflex inhibition may be mediated via mechanoreceptors with low discharge thresholds. Aspiration of an experimentally established effusion causes a biphasic response. An abrupt but incomplete return of function is followed by a slow return of the remaining motor drive (12,46). This pattern suggests that two receptor systems are involved. Again the Type I and II mechanoreceptors are considered to be the active end organs and the necessary stimulus to be alteration of intra-articular pressure.

The joint cavity, as limited by the insertion of the joint capsule, is a container of fixed volume. Intra-articular pressure in normal resting joints is at or slightly below atmospheric pressure (17,26,29). Infusion of a fluid or accumulation of a post injury effusion will result in an increased intra-articular pressure (17,29). Indeed, rheumatoid knees are found to have significantly higher resting pressures than normal knees (29). Jayson and Dixon report that intra-articular pressure is least with the knee positioned at 30° of flexion (29) while Favreau and Laurin report the lowest pressure at 40° of flexion (17). Both groups agree that intra-articular pressure is greater at full extension. Similarly, pressure will increase when the periarticular musculature compresses the capsule as it extends the joint.

Quadriceps inhibition varies with knee joint position. In 1982 Stratford demonstated that inhibition is greater at 0° than 30° of flexion (51). These positions correspond with positions of greater and lesser intra-articular pressure respectively. Favreau and Laurin (17) suggest that rheumatoid knees often develop flexion deformities since this position allows greatest accommodation of fluid with least capsular stress. This

position is also one in which muscular inhibition is least. It is interesting to note that the position of greatest inhibition corresponds to the position of greatest Type I and II endorgan discharge (45). This suggests that capsular stress sets up receptor discharge and muscular inhibition which tends to reduce the capsular stress to a more normal level.

The accumulation of a joint effusion, experimentally induced or following even minor joint injury, will cause a perceptible increase in intra-articular pressure. Type I and II mechanoreceptors signal this change and initiate a tonic inhibition of the knee extensors and facilitation of the flexors. Type II discharge rapidly returns to resting level of activity; however, Type I receptors will continue to fire (45,56). Differing adaptation qualities of the joint endorgans result in a biphasic recovery upon aspiration of a joint effusion.

1.11 TRANSCUTANEOUS ELECTRICAL NERVE STIMULATION

Transcutaneous electrical nerve stimulation is the application of controlled low voltage electrical pulses to the nervous system by passing electricity through the skin

via electrodes placed on the skin (33). Use of electrical stimulation for therapeutic benefit has been dated to 46 AD. when Scribonius Largus used electric eels in treatment of headaches and gout (33). Electric current in various forms has been used since to treat an extremely wide variety of problems. Melzack and Wall's 1965 paper on the gate control theory of pain has led to a revival of the use of electricity as a form of analgesia (55).

In 1978 Mannheimer's group found that transcutaneous electrical nerve stimulation (TENS) significantly increased loading times in 18 of 19 patients with rheumatoid arthritis (32). Recently, use of TENS on injured knees was shown to significantly improve the strength of a maximal voluntary isometric contraction (deSouza, unpublished data). Research has suggested that TENS reduces time to recovery in patients who have undergone knee surgery (30). Typically used for its properties as an analgesic, TENS may also have an effect on reflex inhibition.

Several explanations have been offered as to the possible mechanisms through which TENS is effective. In a 1985 review, Belanger presents the physiological evidence that implicates the endogenous opiate system in TENS

mediated analgesia (5). As early as 1926 A delta and C fibers were identified as those responsible for the transmission of painful stimuli from the nociceptor to the spinal cord (1). These fibers are analogous to Wyke's Group III neurons. C fibers are small diameter, slow conducting unmyelinated neurons. When stimulated under laboratory conditions they produce a diffuse aching sensation that tends to outlast the stimulus. In contrast A delta fibers are slightly larger, thinly myelinated neurons that transmit stimuli interpreted as sharp well localized pain, such as that caused by pinprick or laceration (10,32,53).

Systemic opiates have been noted to inhibit C fiber influence to a greater degree than A delta fibers and inhibit A beta fibers (touch and pressure) the least (19). This would indicate that an opiate mediated analgesia system would be effective in control of pain mediated by C fibers.

Chronic, diffuse, aching sensation carried by C fibers is practically identical to the joint pain reported by patients with rheumatoid arthritis. In Mannheimer's study, TENS-provoked systemic opiates may have resulted in decreased perception of pain and an ability to sustain a contraction in greater comfort and therefore with longer

loading times (32).

Systemic opiates inhibit A beta fibers (touch and pressure) least. Evidence indicates that it is the mechanical stimuli of joint effusion that is the principal cause of reflex inhibition. Such stimuli are delivered to the spinal cord through the A beta fibers. If TENS effectively reduces the experience of reflex inhibition, it must accomplish this through a mechanism other than use of systemic opiates.

Since 1965 the Gate Control Theory of pain has been cited as an explanation for the effectiveness of TENS (55). First proposed by Melzack and Wall this model suggests that peripheral non noxious stimuli may effectively shut down transmission of painful stimuli at the spinal level. Central to this model is the role of a "transmission cell" located in lamina V of the spinal dorsal grey matter. This cell represents the gating mechanism in transmission of noxious stimuli to higher centers. It is proposed that incoming signals from the nociceptive afferents compete with the large diameter fibers to control the gating mechanism. A dynamic equilibrium is established as this incoming information exerts opposing effects on an inhibitory

interneuron located in the substantia gelatinosa. Mechanical stimulation tends to facilitate the activity of the inhibitory cell, while noxious events tend to inhibit the inhibitory interneuron. Whether the gate is opened or closed depends on the predominating input from the two sources. If pain is to reach the conscious level the inhibitory interneuron must be shut down by incoming noxious stimuli. This requires that there be greater input from pain conveying fiber systems.

Use of TENS may cause a preponderance of large fiber activity at the spinal level. This would close the gate to transmission of painful stimuli. However, as noted earlier, pain is not always a necessary stimulus for the production of reflex inhibition. It is not clear if TENS could reduce the experience of inhibition in a non painful joint. Like the endogenous opiate model, the gate control theory gives inadequate insight into the effect of TENS on reflex inhibition.

In a study unrelated to the use of TENS, it was found that cutaneous electrical nerve stimulation can reduce firing thresholds of fast twitch motor units (25). Delwaide and co-workers (13) found that similar stimulation can

increase motor neuron excitability in humans. These findings suggest that TENS may reduce the effect of reflex inhibition by favorably altering anterior horn cell activity.

1.12 CLINICAL CONSIDERATIONS

Reflex inhibition is thought to contribute to quadriceps atrophy, delayed recovery and increased risk of repeated injury (57). Basmajian (4) adds the occurrence of capsular and synovial thickening, effusion and pain to this cycle. Quadriceps inhibition, as great as 80 %, has been measured following joint surgery. Two weeks later inhibition of 50 % persists (42). It is probable that a degree of inhibition persists until the joint effusion resolves.

During rehabilitation, use of imposed motor nerve stimulation (faradic stimulation) has been claimed to assist a patient in overcoming the effects of reflex inhibition (50). Although such stimulation will offer incomplete activation, it will augment voluntary effort. Stratford suggests that the optimal knee joint angle during exercise to be approximately 30° of flexion (51). This position is

one of least intra-articular pressure and minimal inhibitory influence. Cooling the involved joint before and after activity may reduce swelling and pain. Evidence exists that holds promise for the use of TENS; however, its influence on reflex inhibition has not been clearly established.

In summary, the literature reviewed here reveals that there is an articular receptor system in place that is capable of producing prolonged inhibitory influences on selected periarticular muscle groups. This receptor system, functioning in the normal joint, is thought to augment habitual movement patterns. The necessary stimulus for this activity is a change in the intra-articular environment that would logically occur during that movement pattern. In the experimental or injured state the altered joint environment would set up a reflex that leads to depressed extensor activity and facilitated flexor activity.

Although some evidence exists that suggests that TENS may reduce the experience of reflex inhibition, the physiological basis for this is unclear. Despite this lack of insight the clinical significance of these studies remains.

There is evidence which suggests that reflex inhibition is a significant component of joint injury, arthritic processes and post surgical recovery. Further research is warranted in each of these areas. Ideally, such research will include not only a description of the magnitude and duration of inhibition but also an investigation of the efficacy of various treatment techniques.

CHAPTER II

THE INFLUENCE OF ARTHROSCOPY AND POST SURGERY TRANSCUTANEOUS ELECTRICAL NERVE STIMULATION ON QUADRICEPS STRENGTH AND MOTOR UNIT ACTIVATION

2.1 Introduction

Reflex inhibition/facilitation is a frequent and significant consequence of joint trauma, disease and surgical insult. Joint pathology will prevent full activation of certain muscle groups while promoting greater activity in others. It is suggested that injury causes direct inhibition of motorneurons by reflexes which reduce net excitation of motorneurons despite maintained voluntary drive (47).

Reflex activity of the muscles surrounding an injured joint has been documented most frequently following knee injury. Typically a pattern of quadriceps inhibition and normal or facilitated activity of the adductors and knee

flexors is observed (47). The resultant quadriceps atrophy can be expected to delay rehabilitation and render the joint vulnerable to repeated injury, resulting in capsular and synovial thickening, effusion and pain (4). A cycle of injury, reflex inhibition, muscle atrophy, and further injury may thus be established (50).

Shakespeare and associates (19) have examined the magnitude and duration of reflex inhibition following arthrotomy and menisectomy. Using surface EMG techniques, they found that inhibition of the quadriceps was 80 % in the first 24 hours following surgery and this decreased to 73.5 % over the subsequent 3 days. Two weeks post-operative subjects typically demonstrated 30-50 % inhibition of the quadriceps (42).

Clearly, reflex inhibition is a potent complicating event that will occur following arthrotomy and menisectomy. A more benign approach to menisectomy is through arthroscopic intervention. It is reasonable to assume that this less disruptive procedure will cause less inhibition. At present, no data are available describing the magnitude and duration of reflex inhibition that occurs following this popular approach to menisectomy. A major purpose of this

study was to examine the degree of quadriceps inhibition experienced by patients who undergo arthroscopic menisectomy.

Transcutaneous Electrical Nerve Stimulation's (TENS) qualities as an analgesic have been well documented (33). It is not clear if TENS is capable of reducing the phenomenon of reflex inhibition. Mannhiemer and colleagues have demonstrated that TENS significantly increased loading times in 18 of 19 patients with rheumatoid arthritis (32). Recently the use of TENS among a group of patients with injured knees resulted in significant increases in force output during performance of a maximal isometric contraction (deSouza, unpublished data). Jensen and associates (30) reported that TENS significantly reduced the time to recovery of isokinetic power in a group of patients who underwent arthroscopic surgery. Although it is used primarily as an analgesic, TENS may have a role in the relief of reflex inhibition. A secondary purpose of this study was to investigate this role by measuring the change in quadriceps inhibition following a conventional TENS treatment.

METHODS

2.2 Subjects . Subjects were male and female patients (ages 16 to 59 years) who underwent arthroscopic menisectomy in the Hamilton Hospital System between January and November of 1986. Participation in the study was voluntary and subjects received a token honorarium and travel allowance for participation. The project was performed with the approval of the McMaster University Ethics Review Board.

Subjects were tested before surgery and on each of the first 2 days following surgery. Post operative measures were made before and after a 40 minute treatment period. All subjects had the injured and normal leg tested. Thus, 5 complete data sets were collected for each leg. The test schedule is illustrated in Figure 1.

Fifty five patients were referred to the study by 3 area orthopedic surgeons. Admission to the study was based on the criteria listed in Table 1. Twenty six subjects underwent pre surgical testing, (Table 2). Fifteen subjects returned to complete testing on day 1 post surgery. Eleven of the original 26 attended on all 3 test days. Of those who

completed the study, 6 received a true TENS treatment and 5 received a placebo TENS treatment during the treatment period.

2.3 Test Procedure . Measurements were made on a specially constructed device shown in Figure 2. All tests were performed with the knee and hip positioned at 38° and 15° of flexion respectively. To stabilize the leg, a strap was firmly secured over the distal third of the thigh. A second strap, connected at right angles to a force transducer (Sensotec PEP 19824), was positioned over the shin. Impulses received from the force transducer were channelled through a custom made strain gauge amplifier with auto zero capacity. In all instances the shin strap was 28.5 cm from the medial joint line of the knee. In this manner the lever arm was kept constant throughout testing. This allowed measurements of force to be converted to torque values.

Carbon impregnated rubber electrodes, thinly coated with a conductive gel, served as stimulating electrodes. To excite the quadriceps muscle, stimuli were applied to the femoral nerve lateral to the femoral artery. A 10 x 4.5 cm electrode served as a cathode. The anodal electrode, of similar size, was placed over the distal portion of the

quadriceps. Stimuli were rectangular voltage pulses, 500 us in duration, delivered from a stimulator, (model 3072, Digitimer Ltd.).

Maximal voluntary and evoked twitch contractions of the quadriceps were recorded by and stored in a DEC PDP-11/03 microcomputer. Visual display of voluntary and evoked contraction recordings were available on a H.P. Storage Oscilloscope (no.1201B). Printed outputs of each test were collected from a Centronics 150.3 printer. Photographs of interpolated twitch recordings were made with a HP 1978 Camera. The experimental apparatus is depicted in Figure 2. Test procedures in the order presented to the subject are listed in Table 3.

2.4 Resting Twitch Tension . Stimuli of increasing intensity were delivered to the femoral nerve and the resultant twitch contractions observed on an oscilloscope. The point at which further increases in stimulus intensity caused no increase in twitch torque was considered the maximal resting torque tension of the quadriceps. Each subject's resting twitch torque was determined before any other measurements were made. This value was used in statistical analysis.

2.5 Best Maximal Voluntary Contraction . During each test period subjects performed 5 maximal vountary isometric contractions. Patients were instructed to extend their leg against the fixed shin strap and hold a maximal contraction for 3 to 5 seconds. Verbal encouragement was given. The greatest torque developed in any of the 5 maximal voluntary contractions (Best MVC) was selected for statistical analysis.

2.6 Motor Unit Activation . Motor unit activation (MUA) was determined by the twitch interpolation technique, (5,14,34). During a true maximal voluntary contraction of the quadriceps, all of the motor units of that muscle are considered to be fully activated. In this case, no increase in tension can be evoked by an imposed maximal twitch stimulus, (Figure 3a). If the contraction is submaximal, due to the influence of a knee injury, then those motor units not firing, or firing at less than optimal frequency, may be activated by an interpolated maximal twitch stimulus applied to the femoral nerve, and an increment of torque will be observed In this case, a brief increment in torque, the

interpolated twitch (IT), will be observed (Figure 3b). The resting twitch torque (RTT) value and the interpolated twitch value were substituted in the formula given below. The resultant value is an expression of the motor unit activation during that MVC. During each test period, 3 such measures were made. The measure during which greatest motor unit activation (Best MUA) was achieved was selected for statistical analysis.

$$MUA = \frac{RTT - IT}{RTT} \times 100$$

2.7 Visual Analogue Pain Scale . Subjective reports of pain were collected through use of the vertical visual analogue scale (VAS), (37,41). The VAS consists of a vertical line of 100 mm, with each end representative of the extremes of the sensation of pain (Figure 4). Each subject indicated the amount of pain experienced by a horizontal pen mark on the VAS. Each motor unit activation test required the subject to perform an MVC. To gain knowledge of the pain experienced while these tests were performed subjects were required to complete 2 pain scales. The first VAS indicated the amount of pain present at rest. The second VAS indicated

the amount of pain present during the MVC. Pre MVC scores were subtracted from scores representing pain during the MVC. These values were totalled and averaged. The mean score was considered representative of the pain experienced during MVCs. The average pain experienced before a treatment period was compared to the average pain experienced following a treatment period. This information was collected at post operative test sessions, on the injured leg only.

2.8 Transcutaneous Electrical Nerve Stimulation . A Neuromod Selectra (no.7750) TENS unit was used in this study. Those subjects in the treatment group received electrical stimulation of 90 Hz with a 80 microsecond pulse width in a spike waveform. A dual channel criss cross electrode placement technique was used, (Figure 5). Treatment duration was 40 minutes. Intensity was adjusted until the subject reported a firm but comfortable tingling sensation.

Those subjects in the placebo group had 2 electrodes applied on each side of the knee. Leads from these electrodes were inserted into channel 2 of the TENS unit. Channel 1 leads and electrodes were absent. The unit was

turned on and channel 1 intensity adjusted to 10. Channel 2 was electronically silent. The subject observed that the unit was on and the intensity had been adjusted. The placebo condition was maintained for 40 minutes.

2.9 Statistical Analyses Data representing each dependent measure were subjected to separate analyses of variance . Before each result is presented the exact type of ANOVA is reported. Significance was set at p=.05. Each interaction was investigated using a Tukey "a" post hoc statistic.

TABLE 1 INCLUSION and EXCLUSION CRITERIA

Inclusion Criteria

Clinical diagnosis of a meniscal injury later confirmed by arthroscopic surgery and treated by partial or total menisectomy.

Exclusion Criteria

Medial Collateral Ligament Injury

Recent Muscle Injury

Recent Knee Joint Immobilization

Knee Joint Instability

Evidence of Rheumatological Disorders

Evidence of Neuromuscular Disorder

TABLE 2 SUBJECTS

SUBJECT	AGE (yrs.)	INJURED LEG (R / L)	SEX (M / F)
J.D.	20	R	M
J.M.	41	L	M
W.MC.	25	L	F
J.MC.	27	L	M
P.R.	40	R	M
A.E.	31	L	M
A.L.	39	L	M
P.V.	25	R	M
S.W.	21	R	M
B.K.	31	L	M
M.MC.	19	R	M
D.T.	16	L	M
F.D.	36	L	M
K.G.	38	R	M
J.D.	40	R	M
M.A.	32	L	M
M.M.	24	L	M
B.R.	23	R	M
S.D.	19	R	F
M.K.	18	R	F
M.D.	59	R	M
P.W.	19	R	M
F.K.	35	R	M
G.S.	28	R	M
J.P.	26	R	M
M.G.	19	R	F
	$\bar{x}$ = 28.88	R = 16 L = 10	22 M 4 F

TABLE 3 ORDER of TEST PRESENTATION
(Injured Leg Post Surgery)

Resting Twitch Tension
MVC 1
Twitch Tension
MVC 2
Twitch Tension
VAS Ia MVC 3 With Interpolated Twitch VAS Ib
Twitch Tension
VAS IIa MVC 4 With Interpolated Twitch VAS IIb
Twitch Tension
VAS IIIa MVC 5 With Interpolated Twitch VAS IIIb

VAS : Visual Analogue Pain Scale

MVC : Maximal Voluntary Contraction

FIGURE 1.: SCHEDULE OF TESTS AND TREATMENT PERIODS

FIGURE 1. TEST SCHEDULE

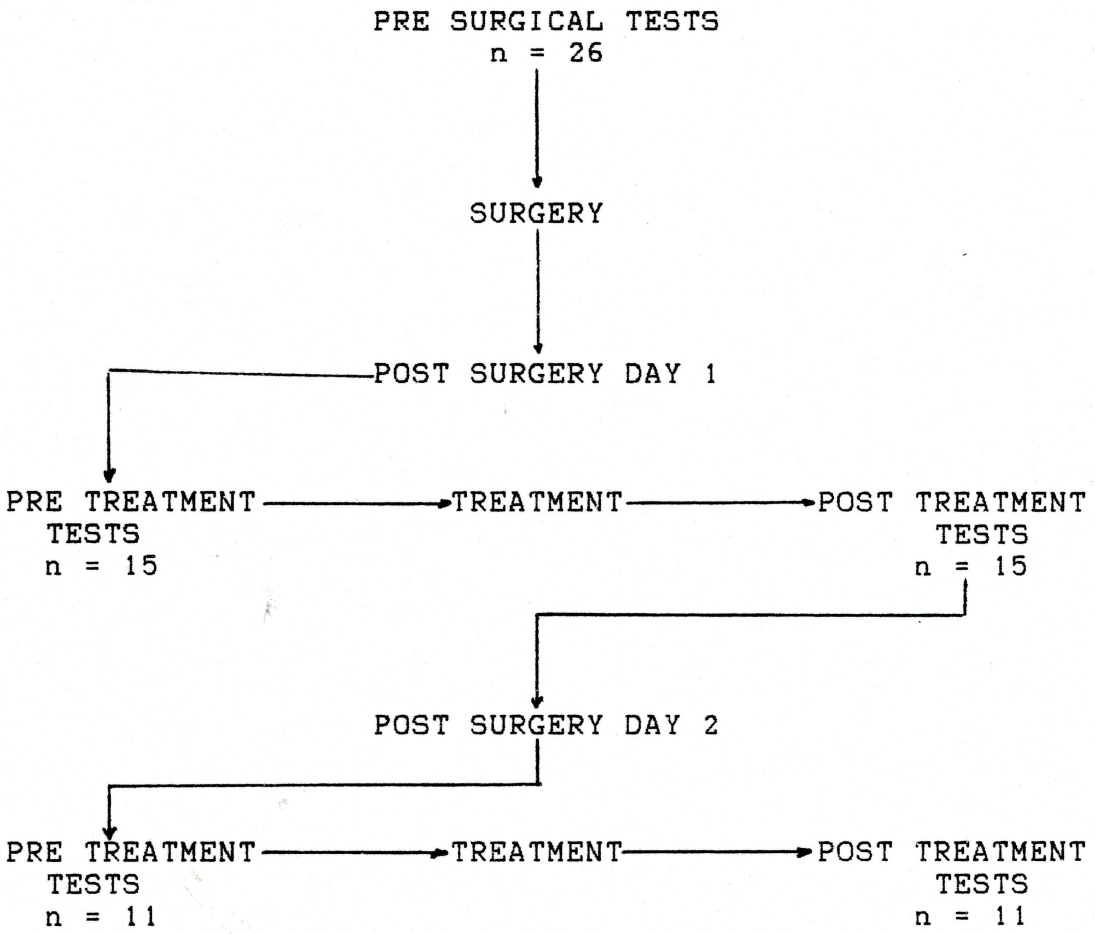


FIGURE 2.: EXPERIMENT APPARATUS

ANGLE OF HIP = 165°

ANGLE OF KNEE = 142°

FIGURE 2: APPARATUS

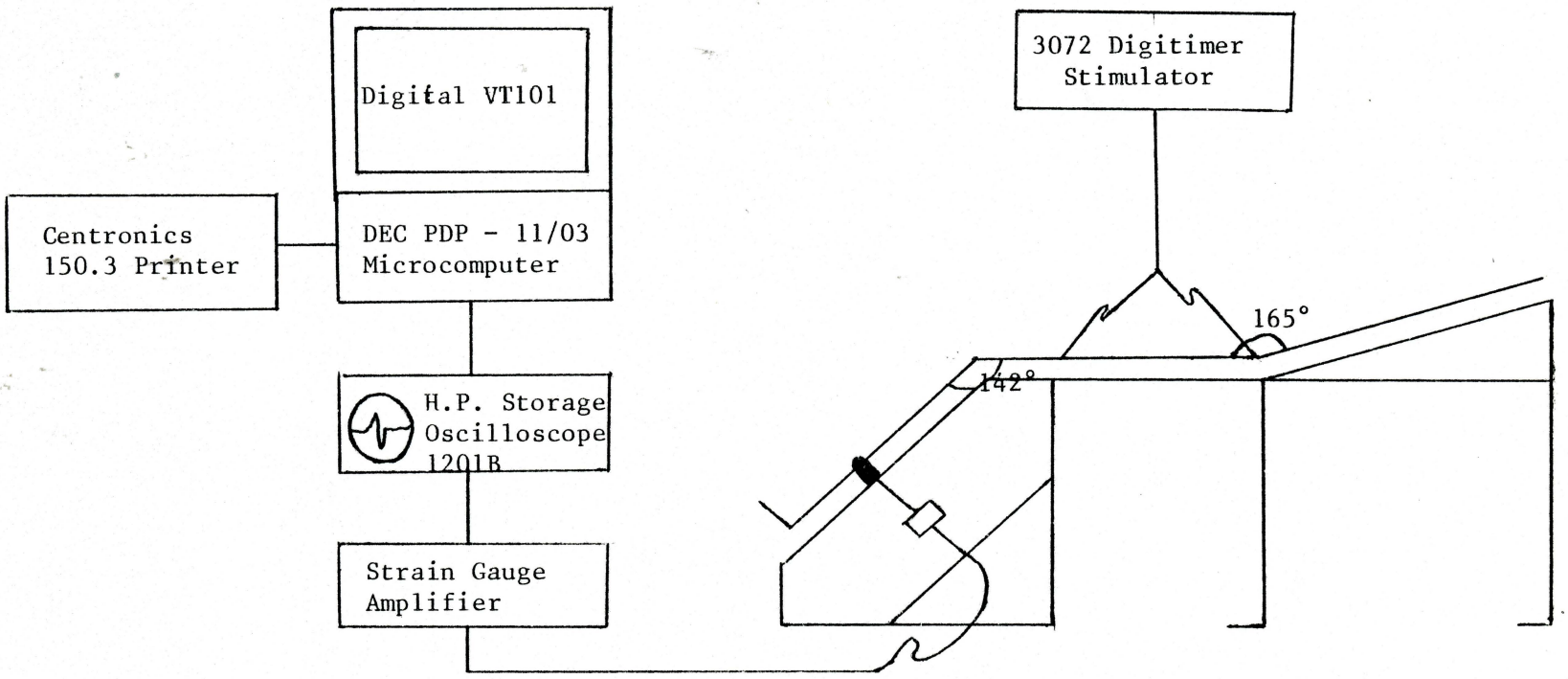


FIGURE 3.: OSCILLOSCOPE DISPLAY OF MVC AND IMPOSED TWITCH TENSION

LOWER CHANNEL : TRACE OF TWITCH TORQUE PRODUCED BY AN INTERPOLATED STIMULUS DELIVERED AT PEAK VOLUNTARY TORQUE

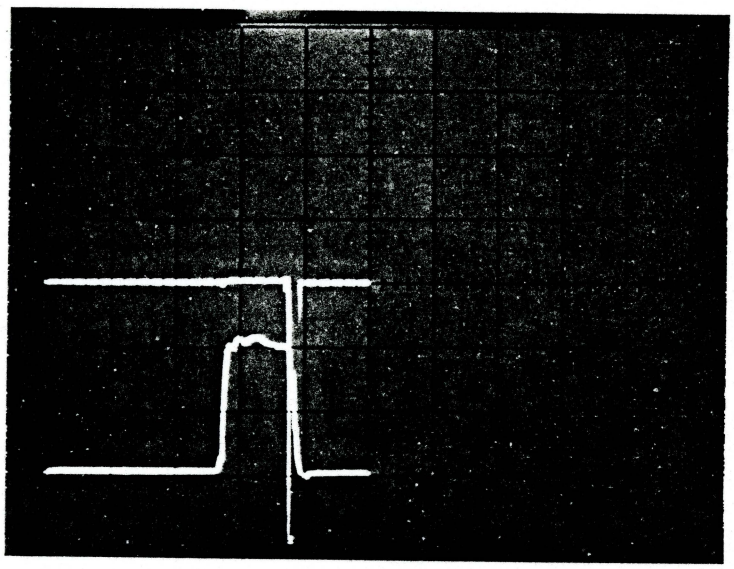
UPPER CHANNEL: TRACE OF MAXIMAL VOLUNTARY ISOMETRIC TORQUE OUTPUT

3A: PHOTOGRAPH DISPLAYING 100 % MOTOR UNIT ACTIVATION

3B: PHOTOGRAPH DISPLAYING 82.1 % MOTOR UNIT ACTIVATION

FIGURE 3.: MOTOR UNIT ACTIVATION

3A : 100 % MUA



3B: 82.1 % MUA

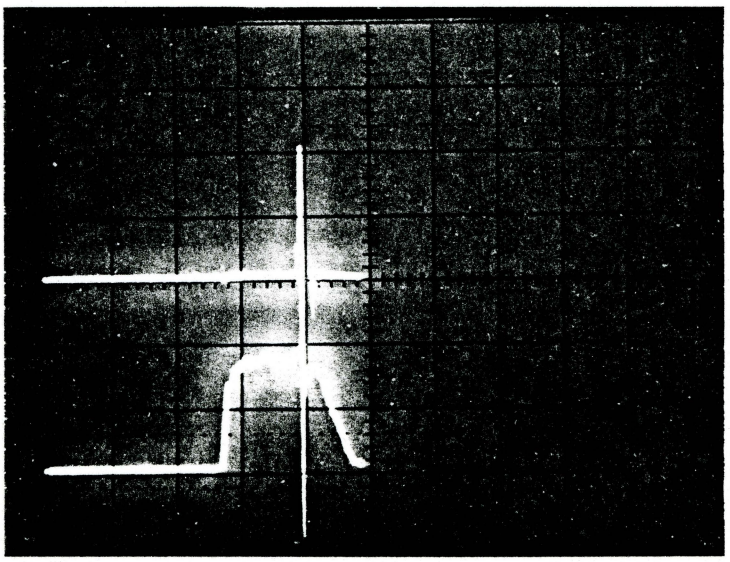


FIGURE 4.: THE VISUAL ANALOGUE PAIN SCALE

The VAS consisted of a vertical line, 100 mm in length. Each end of the line represented the extremes of the sensation of pain. Subjects indicated the amount of pain experienced by placing a horizontal pen mark somwhere along the pain scale.

FIGURE 4: THE VISUAL ANALOGUE PAIN SCALE

Patient:_____ TENS T P

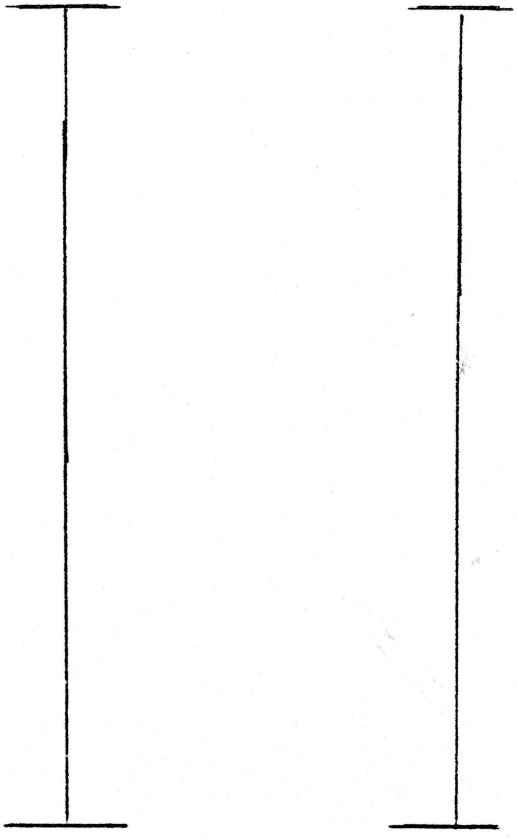
Diagnosis:_____

Date:_____

PRE Rx

Rest 100% MVC

in As Bad As Can Be



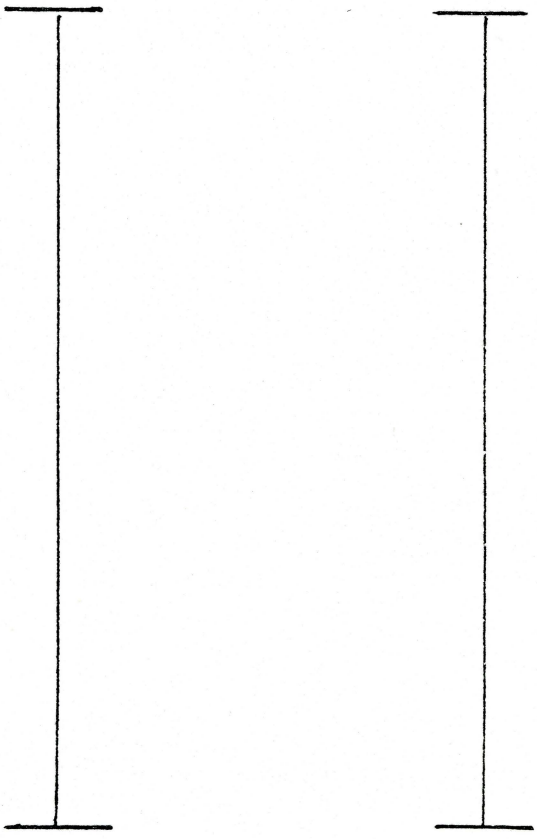
No Pain At all

MVC =

Post Rx

100% MCV

Pain As Bad As Can Be



No Pain At All

MVC =

FIGURE 5.: ELECTRODE PLACEMENT DURING A TRUE TENS TREATMENT

STIMULATION SETTINGS: 90 Hz
80 Microsecond Pulse Width
Spike Waveform

ELECTRODE ARRANGEMENT: Dual Channel
Crisscross Electrode Placement

TREATMENT DURATION: 40 Minutes

FIGURE 5: ELECRODE PLACEMENT DURING A TRUE TENS TREATMENT

Electrode Placement

True TENS

	Channel A		Channel B
A_1	two inches above lateral aspect of patellar base	B_1	two inches above the medial aspect of the patellar base
A_2	below medial condyle of tibia at the level of the tibial tuberosity	B_2	Anterior and inferior to fibular head

2.10 RESULTS

The results were organized into 3 groups for analysis. The first set of data represent the scores of all individuals who attended pre operative evaluations. The second data set consist of measurements taken from subjects who attended pre operative testing and post operative day 1 tests prior to any treatment. The final data include those subjects who attended all five test sessions. Of the 26 people who attended pre operative testing only 11 completed tests on all test days. Reasons for this attrition rate are unclear.

A. PRE OPERATIVE DATA

Twenty six subjects attended pre operative test sessions, (Table 2). All subjects eventually underwent arthroscopic surgery that confirmed a meniscal injury. Data representing each dependent measure were subjected to a one way analysis of variance.

Best MVC . Prior to surgery the injured leg was found to be significantly weaker than the normal leg ($p<.001$). This relationship is demonstrated in Figure 6.

Group means are found in Table 4. Strength values for the injured legs were 23.35 % lower than those for the normal legs.

Motor Unit Activation . Best MUA levels, (figure 7), were significantly lower among injured limbs, ($p<.001$). Comparison of group means (table 4) revealed that MUA levels were 12.31 % lower than scores for the normal leg.

Resting Twitch Torque . Quadriceps resting twitch torque was 13.93 % lower for the injured leg, ($p=.034$), (Figure 8; Table 4)

B. PRE AND POST OPERATIVE DATA

Fifteen subjects completed pre operative and post operative day 1 test sessions. Post operative data used in this analysis were taken before any treatment was administered. These data represent quadriceps performance approximately 24 hours following arthroscopy with medial menisectomy.

Values for each dependent measure were analysed using a two factor repeated measures analysis of variance.

Best MVC Isometric strength scores were significantly reduced following surgery, ($p<.001$). Injured legs were weaker than normal legs before and after surgery, ($p<.001$). An interaction for time and leg was found, ($p<.001$). This interaction indicated that the injured leg underwent a greater decline in performance following surgery than did the normal leg. These relationships are illustrated in Figure 9 and group means are listed in Table 5.

Comparison of group means revealed that strength values for the injured leg were 18.83 % lower than strength values for the normal leg prior to surgery. Following surgery strength values for the injured leg were 45.33 % lower than strength values for the normal leg. When the post operative values of the injured leg were compared to its pre operative scores it was found that the injured leg experienced a 34.01 % reduction in isometric strength. Similar comparisons of normal leg group means determined that the normal leg experienced a 2.01 % reduction in isometric strength.

Best Motor Unit Activation . Motor unit activation levels were significantly lower following surgery, ($p=.02$). Injured legs had lower MUA levels than normal legs before and after surgery, ($p<.001$). An interaction for time and leg was found, ($p=.026$). This interaction indicates that following surgery ability to activate quadriceps declined to a greater degree in the leg that underwent surgery. Figure 10 depicts the decline in MUA levels found in both legs.

Group means (Table 5) were compared in a similar manner as those for best MVC measures. It was found that before surgery MUA levels for the injured leg were 10.06 % lower than activation levels for the normal leg. After arthroscopy this relationship changed such that MUA levels for the injured leg were 22.73 % lower than the normal leg. When the post operative values for the injured leg were compared to its pre operative scores it was found that the injured leg experienced a 15.94 % reduction in activation levels. The normal leg experienced a 1.99 % decline in performance following surgery.

Resting Twitch Tension . Resting twitch tension scores were significantly lower for the injured leg, ($p=.008$). This relationship, (Figure 11), was not

significantly altered following surgery. Comparison of group means (Table 5) established that resting twitch tension values were 23.68 % lower for the injured leg.

C. TRANSCUTANEOUS ELECTRICAL NERVE STIMULATION

Eleven subjects completed all test sessions. True and placebo treatment groups consisted of 6 and 5 subjects respectively. Tests were performed before and after treatment periods on day 1 and 2 post surgery.

Data for each dependent measure were analysed using a 3 factor, mixed design analysis of variance. Interactions were subjected to post hoc (Tukey "a") analysis.

Analysis determined that neither true nor placebo treatments had any effect on strength or motor unit activation. F values for the necessary 3 way interactions were F=.682 for best MVC data, and F=.934 for best MUA data.

Best MVC Following surgery, strength was significantly less in both the injured and normal leg,

($p<.001$). Injured legs were weaker than normal legs at all times of test ($p=.001$). An interaction for time and leg was found ($p=.011$). This indicates that there was a greater loss of strength in the injured leg following surgery. True or placebo TENS had no effect on performance of the strength test (Figure 12).

Mean values are listed in Table 6 for the injured and normal legs at each time of test. For the injured leg, it was found that each post operative mean was lower than the pre operative value, ($p=.01$). Means at time 1A and 2B were compared to determine if any recovery of strength had occurred from day 1 to day 2 post surgery. Although mean strength scores were higher on day 2 the difference was not significant. Since scores at times 1A and 1B and times 2A and 2B did not vary significantly they were averaged in order that pre surgical performance could be compared to performance on day 1 and day 2 post surgery. In this manner it was determined that day 1 and day 2 strength scores were 29.3 % and 21.05 % lower than pre surgical measures.

Strength scores for the normal leg at times 1A, 2A and 2B were similar to that legs pre surgical performance.

At time 1B the strength value was found to be significantly lower than the pre surgical mean, ($p=.05$).

Best Motor Unit Activation Motor unit activation (MUA) levels were reduced following surgery, ($p<.001$). At all times the injured leg had lower MUA levels than the normal leg ($p=.003$). Following surgery the injured leg demonstrated a greater loss of MUA than the normal leg, ($p=.016$). True or placebo TENS had no effect on quadriceps MUA levels. These findings are illustrated in Figure 13.

Table 6 lists the means for the injured and normal leg at each time of testing. Analysis of the means for the injured leg determined that on day 1 post surgery there was a significant reduction in MUA, ($p=.01$). By day 2 post surgery the MUA levels were similar to pre operative values. A significant recovery of MUA was recorded from time 1A to 2A and time 1A to 2B, ($p=.01$). Since MUA values at times 1A and 1B and times 2A and 2B did not vary significantly they were averaged in order that pre surgical performance could be compared to performance on day 1 and day 2 post surgery. Thus it was found that MUA levels for the injured leg dropped from a pre surgical value of 83.06 % to 67.01 % on

day 1 post surgery and climbed to 77.21 % on day 2 post surgery.

MUA levels for the normal leg did not vary significantly from the pre surgical level.

Visual Analogue Pain Scale Subjective reports of pain were reduced to single mean values representing pain experienced while performing a maximal isometric contraction (Figure 14). Scores were collected before and after treatment periods on day 1 and 2 post surgery. Data were analysed using a 3 factor, mixed design analysis of variance.

Type of treatment had no effect on VAS scores. Thus the placebo was as effective as the true TENS treatment protocol. Patients experienced significantly less pain during the second day of testing, ($p=.04$). An interaction for day and time of testing was found. Post hoc analysis indicated that subjects experienced less pain following treatment on day 1, ($p=.05$).

Resting Twitch Tension Analysis of resting twitch tension values revealed that there was no significant difference in any of the collected data sets. A trend was identified in the data for treatment group and leg. Thus twitch tension values tended to be lower among subjects allocated to the true treatment group. Injured legs tended to have lower resting twitch tension values than the normal leg. These relationships are depicted in Figure 15.

TABLE 4 PRE SURGICAL GROUP MEANS

Values represent quadriceps performance approximately 24 hours prior to arthroscopic surgery.

n = 26
Best MVC : Best Maximal Voluntary Contraction
Best MUA : Best Motor Unit Activation
RTT : Resting Twitch Torque

A. BEST MVC (N·m Torque)

	Normal Leg	Injured Leg
$\bar{x}$ (SD)	191.33 (+/- 49.13)	146.67 (+/- 52.92)

B. BEST MUA (%)

	Normal Leg	Injured Leg
$\bar{x}$ (SD)	89.58 (+/- 10.05)	78.56 (+/- 17.58)

C. RESTING TWITCH TENSION (N·m Torque)

	Normal Leg	Injured Leg
$\bar{x}$ (SD)	39.71 (+/- 15.65)	34.16 (+/- 14.66)

TABLE 5 PRE SURGICAL POST SURGICAL GROUP MEANS

Values represent quadriceps performance approximately 24 hours prior to and 24 hours following arthroscopic surgery

n = 15
Best MVC = Best Maximal Voluntary Contraction
Best MUA = Best Motor Unit Activation
RTT = Resting Twitch Torque

A. BEST MVC ($\bar{x}$ Nm Torque +/- SD)

	Pre Surgery	Post Surgery Day 1
Normal Leg	190.55 (+/- 43.92)	186.72 (+/- 45.31)
Injured Leg	154.68 (+/- 52.36)	102.08 (+/- 44.16)

B. BEST MUA ($\bar{x}$ % Activation +/- SD)

	Pre Surgery	Post Surgery Day 1
Normal Leg	90.12 (+/- 11.50)	88.13 (+/- 11.51)
Injured Leg	81.06 (+/- 18.31)	68.10 (+/- 16.15)

C. RESTING TWITCH TENSION ($\bar{x}$ Nm Torque +/- SD)

	Pre Surgery	Post Surgery Day 1
Normal Leg	40.61 (+/- 16.36)	39.58 (+/- 14.26)
Injured Leg	30.92 (+/- 12.25)	29.74 (+/- 13.66)

TABLE 6 TENS DATA, GROUP MEANS

PSx = Pre Surgery
1A = Day 1 Post Surgery, Pre Treatment
1B = Day 1 Post Surgery, Post Treatment
2A = Day 2 Post Surgery, Pre Treatment
2B = Day 2 Post Surgery, Post Treatment

n = 6

A. BEST MVC ($\bar{x}$ Nm Torque +/- SD)

	Normal Leg	Injured Leg
Pre Surgery	196.02 (+/- 40.15)	154.88 (+/- 53.69)
Day 1 Pre Treat.	187.38 (+/- 43.78)	105.97 (+/- 45.33)
Day 1 Post Treat.	174.67 (+/- 47.24)	113.06 (+/- 53.83)
Day 2 Pre Treat.	177.89 (+/- 51.82)	120.05 (+/- 45.44)
Day 2 Post Treat.	176.52 (+/- 45.88)	124.51 (+/- 49.46)

B. BEST MUA (x % Activation +/- SD)

	Normal Leg	Injured Leg
Pre Surgery	91.70 (+/- 7.12)	83.06 (+/- 16.83)
Day 1 Pre Treat.	87.29 (+/- 12.37)	66.25 (+/- 18.22)
Day 1 Post Treat.	84.41 (+/- 12.03)	67.78 (+/- 25.25)
Day 2 Pre Treat.	87.92 (+/- 10.70)	76.87 (+/- 16.49)
Day 2 Post Treat.	86.49 (+/- 7.38)	77.67 (+/- 16.40)

FIGURE 6.: BEST MVC OF NORMAL AND INJURED LEG BEFORE ARTHROSCOPIC MENISECTOMY

n = 26
* p < .001

Data is representative of quadriceps performance approximately 24 hours prior to arthroscopic surgery.

Prior to surgery injured limbs had significantly lower MVC values than normal limbs.

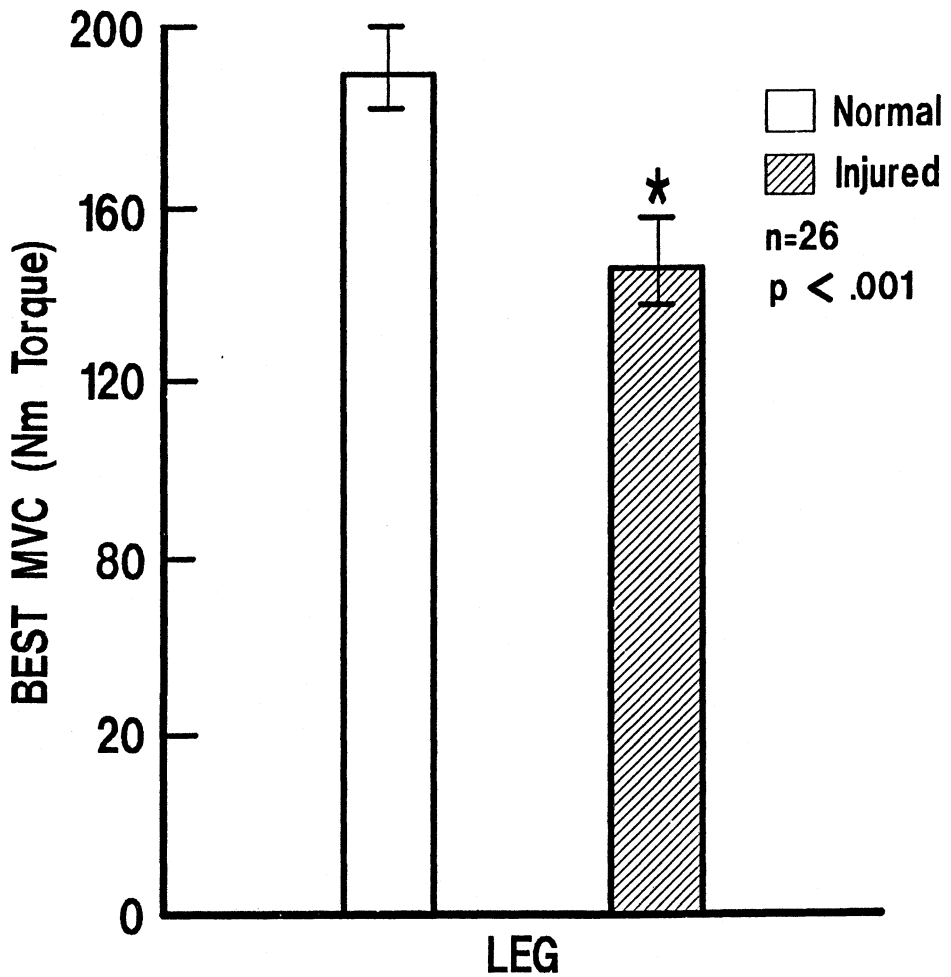


Fig. 6 Best MVC of normal and injured leg before arthroscopic menisectomy.

FIGURE 7.: BEST MUA OF NORMAL AND INJURED LEG BEFORE ARTHROSCOPIC MENISECTOMY

n = 26
* $p < .001$

Data is representative of quadriceps performance approximately 24 hours prior to arthroscopic surgery.

Prior to surgery injured limbs had significantly lower MUA values than normal limbs.

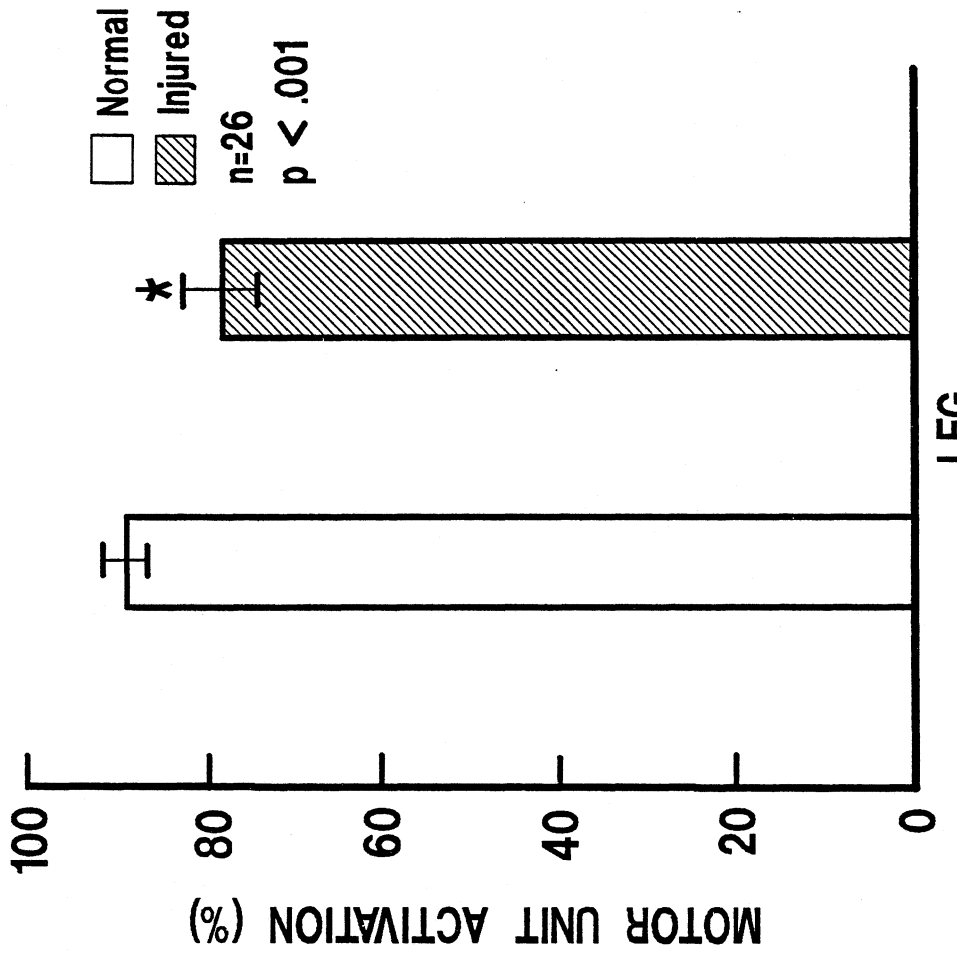


Fig. 7 Best MUA of normal and injured leg before arthroscopic menisectomy.

FIGURE 8.: RESTING TWITCH TENSION OF NORMAL AND INJURED LEG BEFORE ARTHROSCOPIC MENISECTOMY

n = 26
* $p < .034$

Data is representative of quadriceps performance approximately 24 hours prior to arthroscopic surgery.

Prior to surgery the injured limb presented with significantly reduced resting twitch tension values.

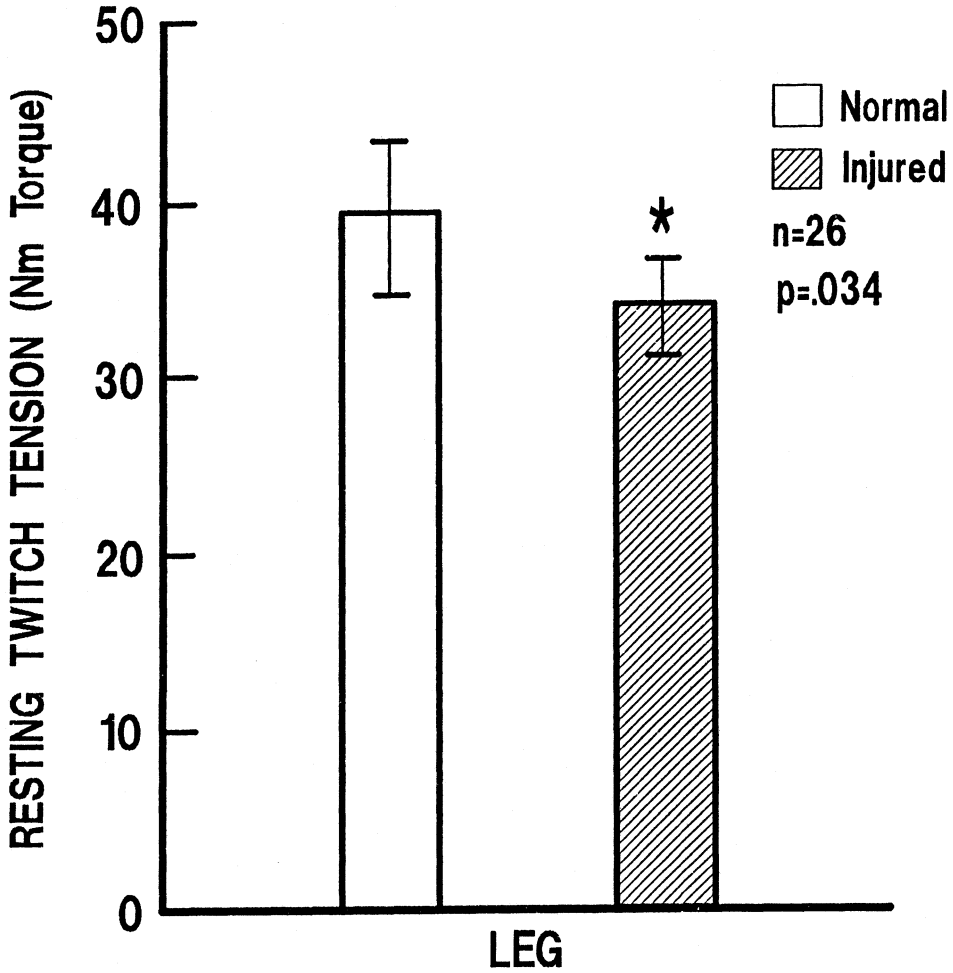


Fig. 8 Resting Twitch Tension of normal and injured leg before arthroscopic menisectomy.

FIGURE 9.: BEST MVC OF NORMAL AND INJURED LEG BEFORE AND AFTER ARTHROSCOPIC MENISECTOMY

n = 15
* p = .001
+ p = .001

Data is representative of quadriceps performance approximately 24 hours prior to and 24 hours following arthroscopic surgery.

Injured legs were weaker than normal legs before and after surgery (*).

The injured leg underwent a greater decline in strength following surgery than did the normal leg (+).

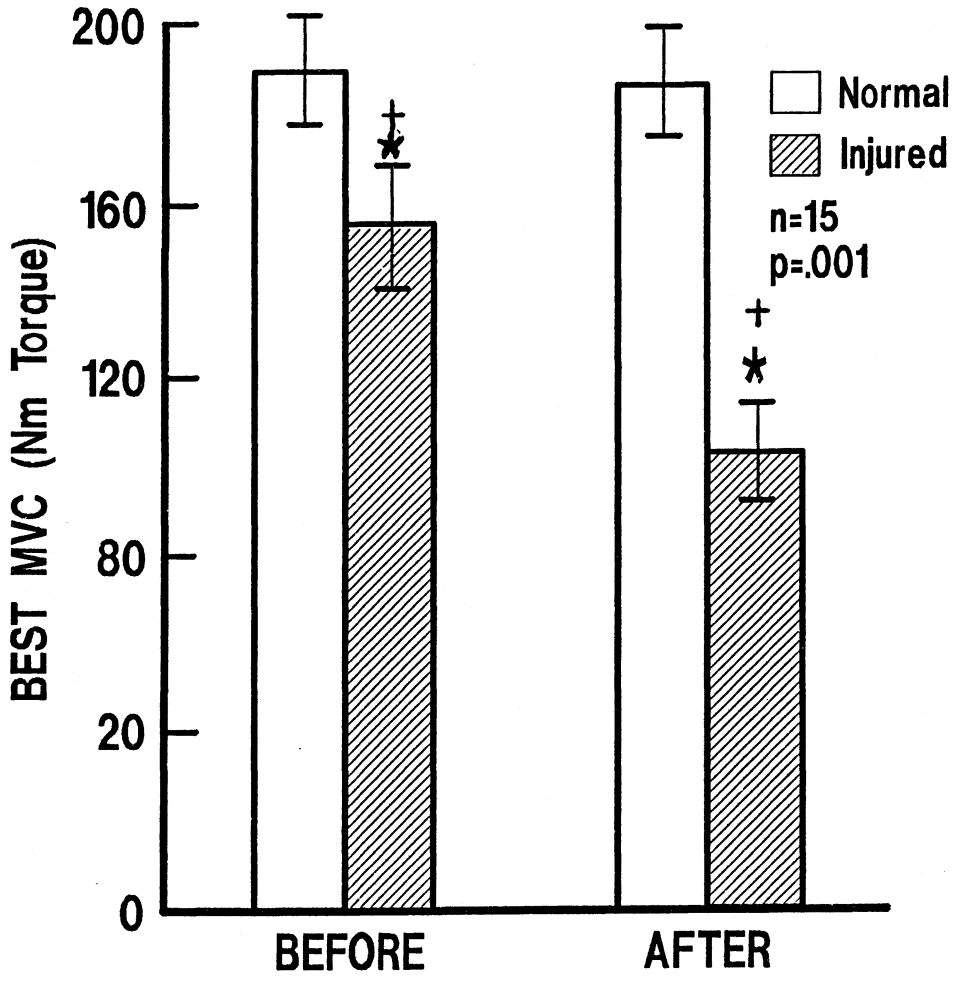


Fig. 9 Best MVC of normal and injured leg before and after arthroscopic menisectomy.

FIGURE 10.: BEST MUA OF NORMAL AND INJURED LEG BEFORE AND AFTER ARTHROSCOPIC MENISECTOMY

n = 15
* p < .001
+ p < .02

Data is representative of quadriceps performance approximately 24 hours prior to and 24 hours following arthroscopic surgery.

Injured legs had lower activation levels than normal legs before and after surgery (+).

Following surgery ability to activate quadriceps declined to a greater degree in the leg that underwent surgery (*).

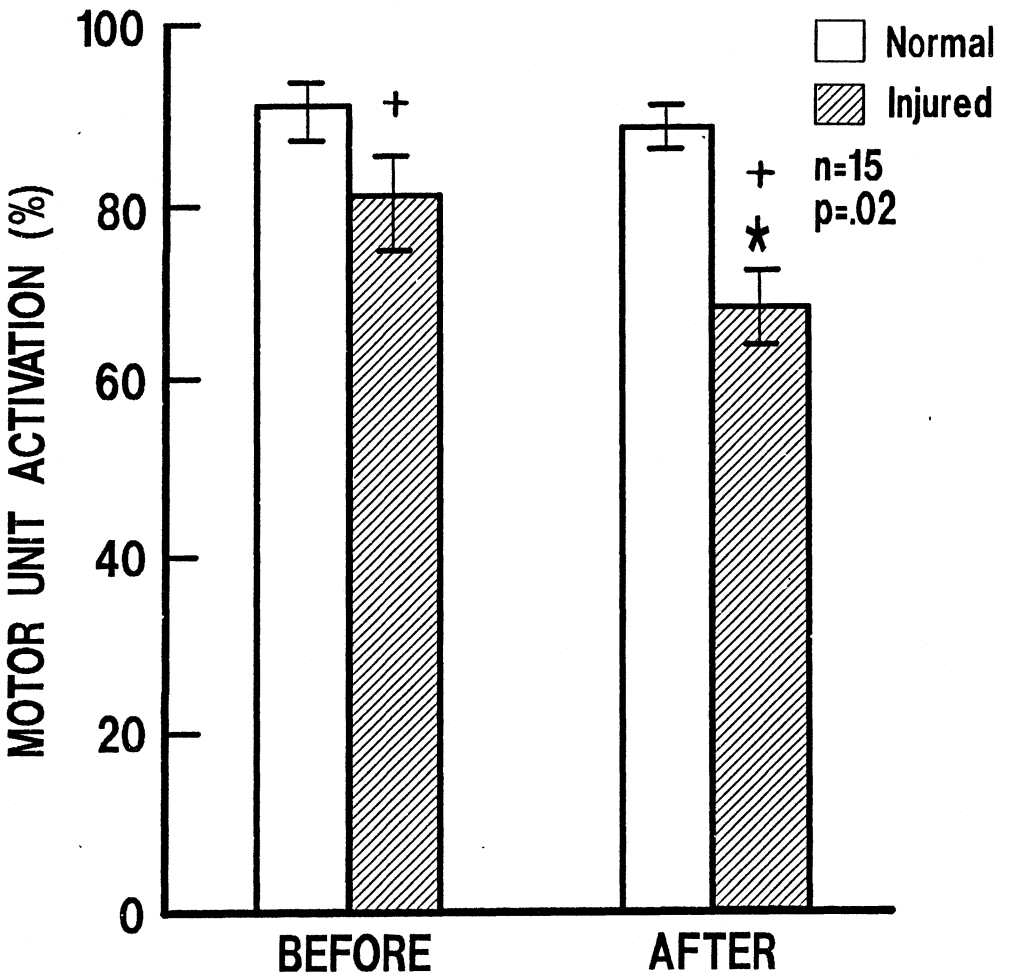


Fig. 10 Best MUA of normal and injured leg before and after arthroscopic menisectomy.

FIGURE 11.: RESTING TWITCH TENSION OF NORMAL AND INJURED LEG BEFORE AND AFTER ARTHROSCOPIC MENISECTOMY

n = 15
* p = .01

Data represent quadriceps performance approximately 24 hours prior to and 24 hours following arthroscopic surgery.

Resting twitch tension values were significantly lower for the injured leg (*). This relationship was not significantly altered following surgery.

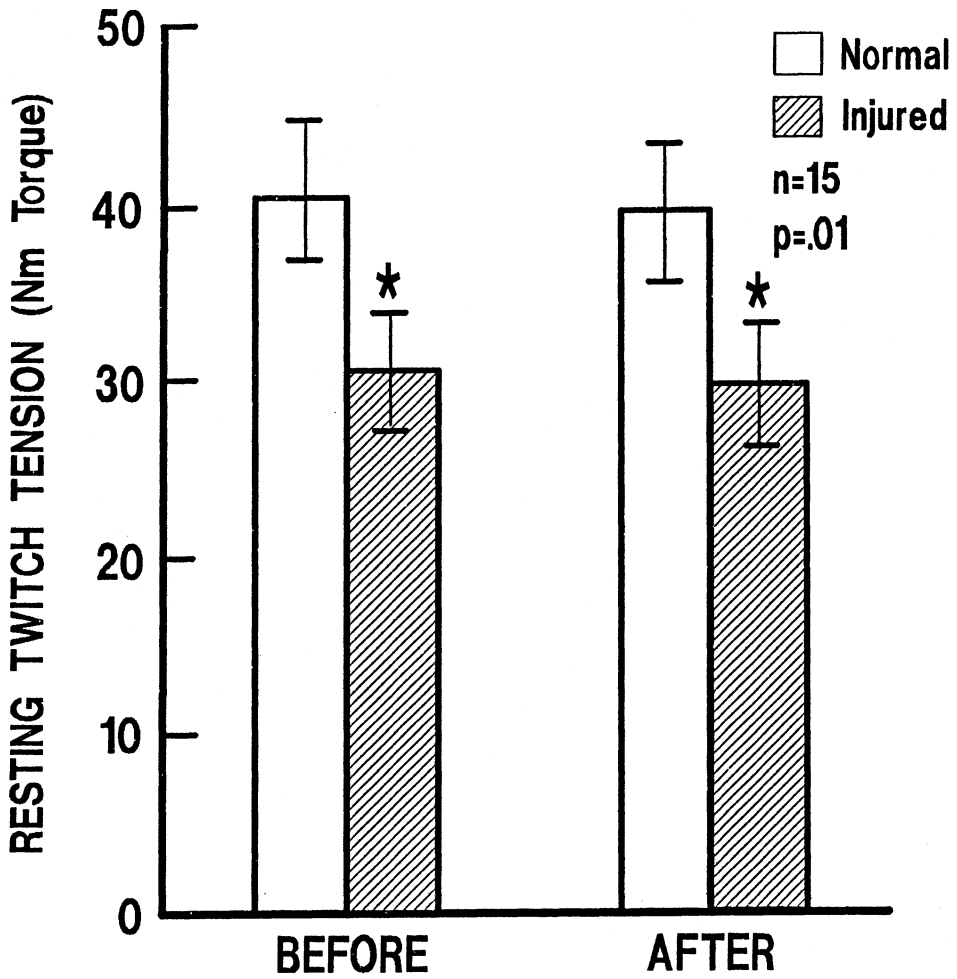


Fig. 11 Resting Twitch Tension of normal and injured leg before and after arthroscopic menisectomy.

FIGURE 12.: BEST MVC OF NORMAL AND INJURED LEG PRE SURGERY AND ON DAY ONE AND TWO POST SURGERY BEFORE AND AFTER TREATMENT

n = 11
+ p = .011
* p = .001

Isometric strength tests revealed that injured legs were weaker than normal legs at all times of testing (*). The injured leg experienced a greater loss of strength than the normal leg following surgery (+). Treatment had no significant effect on quadriceps strength.

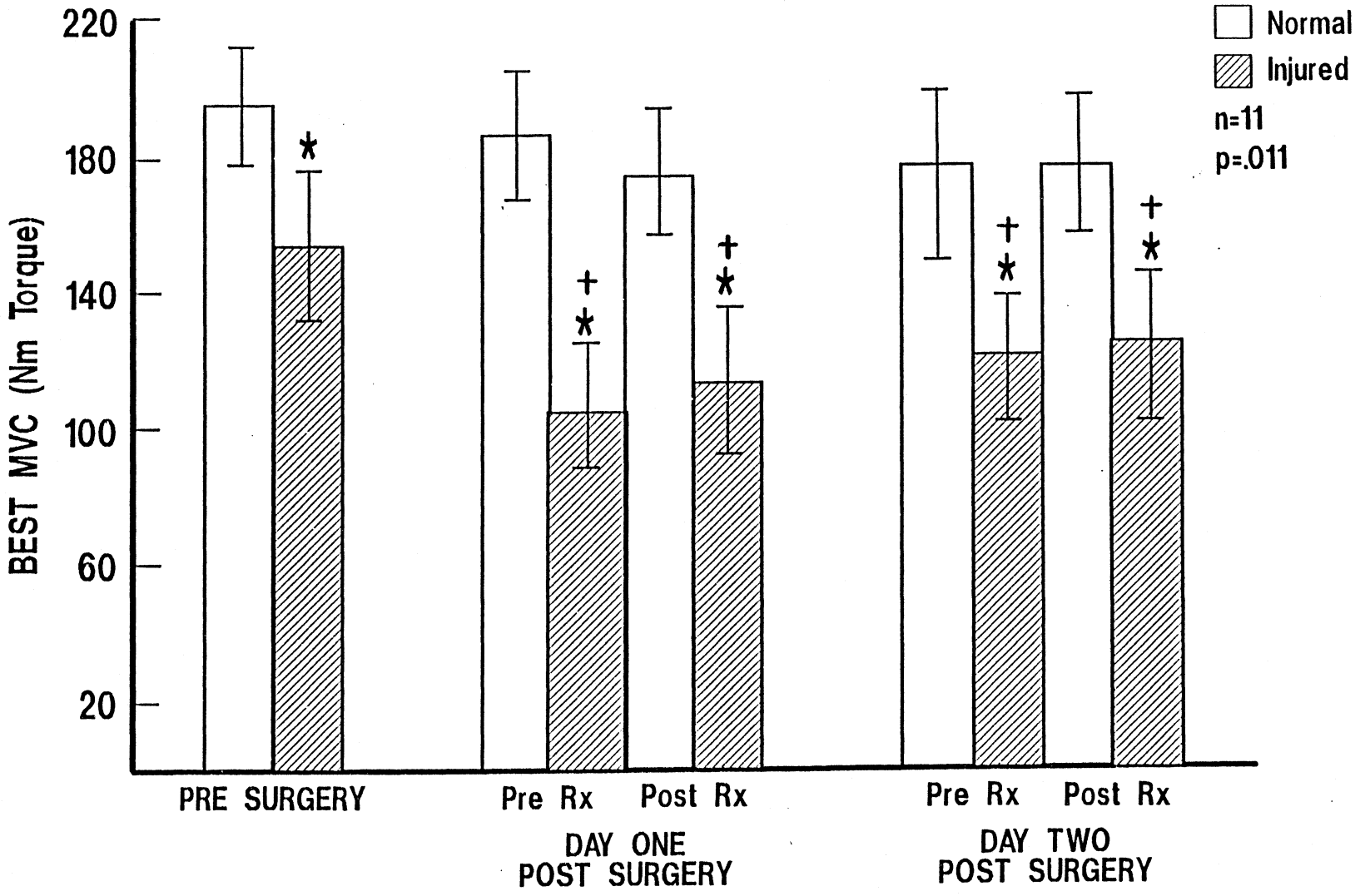


Fig. 12 Best MVC of normal and injured leg pre surgery and on day one and two post surgery before and after treatment.

FIGURE 13.: BEST MUA OF NORMAL AND INJURED LEG PRE SURGERY AND ON DAY ONE AND TWO POST SURGERY BEFORE AND AFTER TREATMENT

n = 11
* p = .016

Injured legs had significantly lower activation levels than normal legs on day one post surgery (*). By day two post surgery, injured leg activation levels had recovered to levels similar to pre surgical values. Treatment had no effect on quadriceps motor unit activation.

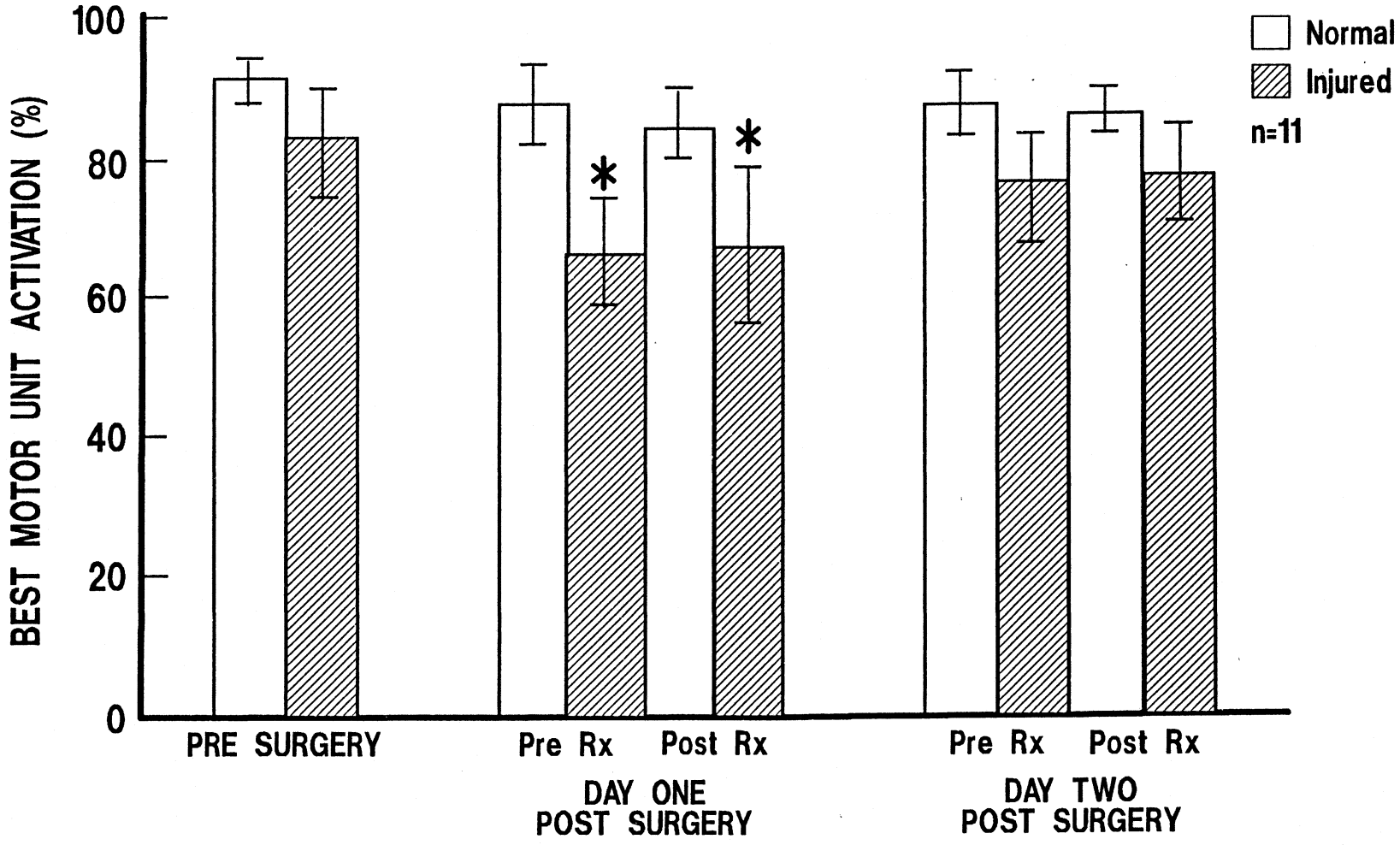


Fig. 13 Best MUA of normal and injured leg pre surgery and on day one and two post surgery before and after treatment.

FIGURE 14.: SUBJECTIVE EXPERIENCE OF PAIN WHILE PERFORMING A MAXIMAL VOLUNTARY ISOMETRIC CONTRACTION: BEFORE AND AFTER TREATMENT

n = 11
* p = .012

Subjects reported significantly less pain while performing maximal voluntary contraction on the second post surgical day (*). Treatment had no effect on the amount of pain reported.

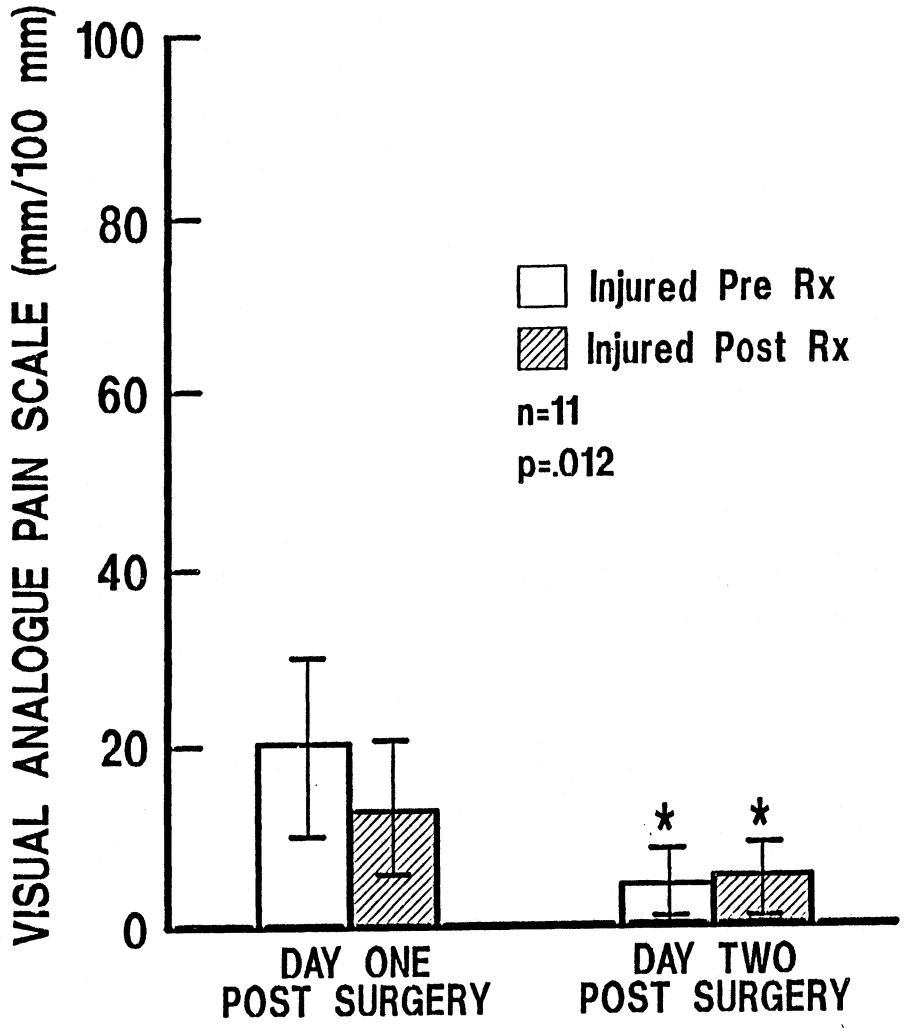


Fig. 14 Subjective experience of pain while performing a maximal voluntary isometric contraction: before and after treatment.

FIGURE 15.: RESTING TWITCH TENSION OF NORMAL AND INJURED LEG PRE SURGERY AND ON DAY ONE AND TWO POST SURGERY BEFORE AND AFTER TREATMENT

n = 11

Injured legs tended to have lower resting twitch tension values than the normal legs. This difference was not significant.

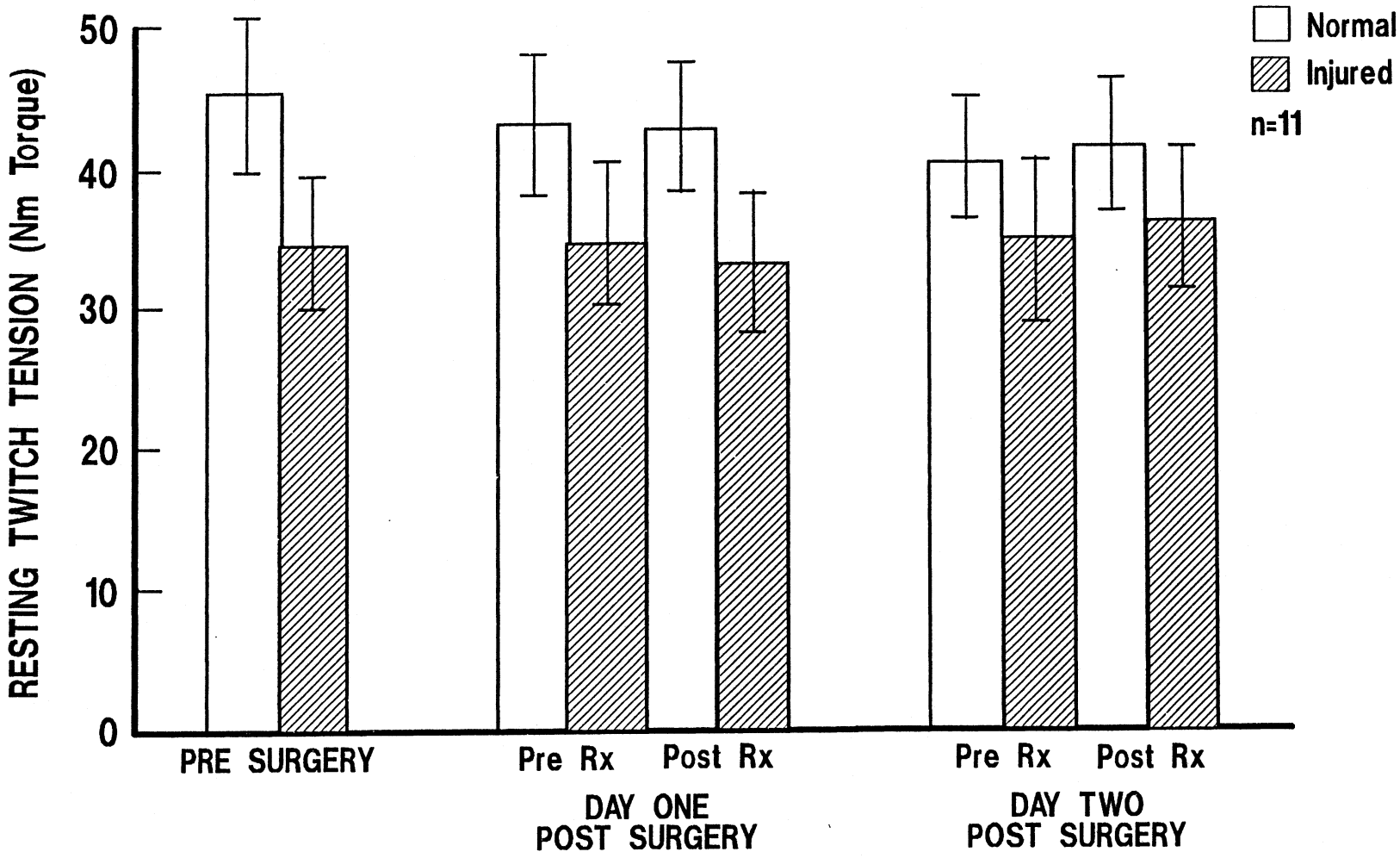


Fig. 15 Resting Twitch Tension of normal and injured leg pre surgery and on day one and two post surgery before and after treatment.

2.12 DISCUSSION

Pre Operative Data

Results of this study indicate that patients awaiting arthroscopy present with significantly reduced quadriceps strength (Fig.6). Strength values for the injured leg were 23 % lower than those for the normal leg. Also, subjects were found to have significantly reduced motor unit activation and resting twitch torques (Fig.7-8). This suggests that, prior to surgery, strength loss is a product of altered neural drive and reduced muscle force generating capacity.

Time between initial injury and date of surgery varied greatly among subjects tested. This interval ranged from several weeks to several years. None of the tested subjects had undergone joint immobilization. Subjects performed their usual daily activities, avoiding only those tasks which aggravated their injury. These factors make it difficult to estimate the role of disuse atrophy in production of strength loss. It is probable that the existence of chronic quadriceps inhibition will have

contributed to the process of muscle atrophy. Young and co-workers have suggested that a cyclical pattern of joint injury, reflex inhibition, muscle atrophy and further injury is established (57).

Patients awaiting knee surgery may benefit from decreased intervals between time of injury and date of surgery. Inhibition and muscle atrophy may be reduced by pre-surgical rehabilitation. Theoretically, this would diminish the length and intensity of post surgical rehabilitation.

Pre and Post Operative Data

Evaluation of quadriceps strength approximately 24 hours following surgery determined that a dramatic decline in voluntary strength had occurred (Fig.9). The injured leg demonstrated a 34 % reduction in voluntary strength while that of the normal leg was unchanged. Resting twitch torque for both legs was similar to pre operative values (Fig.11).

The short interval (approximately 48 hours) between tests makes it unlikely that strength loss is a result of lost contractile tissue or altered muscle qualities. If

such changes had occurred, they would have been reflected in the evoked properties of the muscle. No such changes were observed. Strength loss due to the lingering effects of a general anaesthetic is also unlikely since, if this were the case, the normal leg would have exhibited a similar decrement. Some authors have suggested that use of a tourniquet during surgery may contribute to altered muscle function (15,40). Stokes and associates found tourniquet use to have no effect on muscle function in normal volunteers (50). The finding of unchanged resting twitch values implies that motor units were not adversely affected by surgical technique or anaesthetic and implies the involvement of more proximal components of the neuromuscular system.

Subjects experienced a 16 % decline in motor unit activation levels following surgery in their injured leg (Fig. 10). Prior to surgery, the injured leg already demonstrated depressed activation levels. When this is considered, it is noted that post surgical performance of the injured leg was 23 % lower than that of the normal leg. Since activation levels for the normal leg were unchanged following surgery, reduced neural drive seems to have been a result of surgical events at the injured leg. This is

consistent with the views of several authors who credit intra articular events for the establishment of reflex inhibition (8,12,26,28,42,43,44,46,50,51).

In 1982 Shakespeare and associates found that quadriceps inhibition, as determined by EMG techniques, averaged 80 % in the first day following menisectomy through arthrotomy (42). Shakespeare and co-workers describe inhibition as a percent reduction of pre surgical rectified integrated EMG activity during a maximal voluntary isometric contraction. Due to differences in measurement technique direct comparisons with the present study are not possible. Arthroscopy is considered to be a relatively benign approach to menisectomy when compared to arthrotomy. Thus it is reasonable to assume that arthroscopy would result in less muscular disability. Although this study would tend to support such an argument, further investigation is warranted.

Transcutaneous Electrical Nerve Stimulation

Of those 11 subjects who completed all phases of testing there was observed a 30 % and a 21 % reduction of quadriceps strength on post surgical days 1 and 2

respectively (Fig.12). Although strength improved by 8 % between the first and second day post surgery, this change was not statistically significant. Motor unit activation levels followed a very similar pattern of decline and recovery (Fig.13). In this case, however, recovery was significant between day 1 and 2 post surgery. This suggests that the trend to strength recovery is related to improved neural activation of the muscle tissue. This pattern is regarded as evidence that reflex inhibition is greatest in the first day following surgery. Thereafter recovery to pre surgical levels of muscle inhibition occurs relatively quickly. It is unclear how long reflex inhibition persists following joint injury or surgery.

Measures made on the normal leg revealed that strength following a treatment period on day 1 post surgery was significantly lower than pre surgical values. Reasons for this are unclear.

In review of the history of their knee injury few patients complained that pain was a major feature. Typically, complaints of weakness, perceived instability, joint locking and occasional gross joint effusions were made. Several subjects commented that their knee did not

feel badly enough to warrant surgery. Visual analogue pain scale scores indicate that subjects experienced very little pain following surgery (Fig.14). Furthermore, subjects were able to perform maximal voluntary contractions with very little pain. These reports are not consistent with the degree of disability measured in this study. Significantly altered muscle function may occur following joint injury and surgery even though pain is not a major feature of the condition. This finding is in agreement with several other reports (8,12,42,46,50,51).

Neither strength nor motor unit activation were altered by the TENS treatment. Use of this modality was investigated based on findings of two previous studies. In 1978, Mannheimer and associates determined that TENS improved loading times in 18 of 19 patients with rheumatoid arthritis (32). In 1985 deSouza found that TENS improved the performance of an isometric strength test in subjects suffering from retropatellar knee pain (unpublished data). In both of these studies the major feature of the clinical condition is joint pain. TENS effectiveness as an analgesic is well documented (31). It is conceivable that following TENS treatment the experience of pain was reduced and vigorous exercise became more comfortable and therefore

greater strength output was possible. As already noted pain was not a great problem for the patients tested in the present study. In light of this finding it is not surprising that a modality known for its analgesic properties had no role in relief of post arthroscopic inhibition.

2.13 SUMMARY

The primary goal of this study was to determine the magnitude of quadriceps inhibition and strength loss experienced by patients who have undergone arthroscopic menisectomy. A secondary goal of this study was to determine the influence of Transcutaneous Electrical Nerve Stimulation on the extent of reflex inhibition.

Based on the results of this study the following conclusions may be made:

1. Patients awaiting arthroscopic surgery typically present with a reduction in motor unit activition, a reduction in quadriceps voluntary strength and a reduction in evoked contraction strength.

2. Following arthroscopic surgery both motor unit activation and voluntary strength are further reduced whereas evoked contraction strength remains unchanged in the injured leg.

3. By the second post surgical day voluntary strength remains depressed however a significant recovery of MUA occurs.

4. Patients report little discomfort following arthroscopic menisectomy.

5. Conventional TENS had no effect on voluntary strength or MUA following arthroscopic menisectomy.

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APPENDIX I

THE EFFECT OF TRANSCUTANEOUS ELECTRICAL NERVE STIMULATION ON MAXIMAL STRENGTH TESTING OF PAINFUL KNEES

A PILOT STUDY

THE EFFECT OF TRANSCUTANEOUS ELECTRICAL NERVE STIMULATION ON MAXIMAL STRENGTH TESTING OF PAINFUL KNEES

Key Words: Electroanalgesia, knee joint, isometric contraction

by

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ABSTRACT

The purpose of the present study was to determine the effect of transcutaneous electrical nerve stimulation (TENS) on the performance of an isometric knee extension manoeuver by people with painful knees. In a crossover design, twelve subjects received a true and a placebo TENS treatment. Subjects' performance of a pre and post treatment maximal isometric contraction (MVC) was evaluated as well as their subjective reports of pain according to a visual analogue pain scale (VAS). Although subjects demonstrated a significant improvement in MVC scores following a true TENS treatment ($p=0.009$), no improvement was observed following the placebo treatment. Both the true and placebo treatments significantly reduced the patients subjective report of pain ($p=0.027$). Correlational analysis revealed that there was no relationship between pain relief and amount of force exerted. These results suggest that for patients with painful knees TENS can effectively improve performance of an isometric knee exercise.

INTRODUCTION

An individual's failure to fully activate a muscle group acting about an injured joint has often been attributed to the presence of painful stimuli in that joint (1-3). The usual practice therefore has been to administer modalities designed to reduce pain prior to exercise. Transcutaneous Electrical Nerve Stimulation (TENS) is one such modality. The rationale for using this modality is that at the very least, the injured patient may exercise in greater comfort. Whether the application of TENS actually improves the patient's performance of a prescribed exercise is unknown.

It has been established that inhibition following knee injury may result from stimuli other than pain (4-7). Afferent stimuli related to joint pressure, angle of joint function and synovial congestion have been suggested as possible sources of reflex inhibition. Although inhibition may be overcome through intra-articular administration of a

local anaesthetic (5), this approach, while effective, is not practical due to its short lived effect and invasive nature. In contrast, a modality such as TENS offers the advantages of safety, convenience and ease of application, if its effectiveness can be demonstrated.

The purpose of this pilot study was to determine the effect TENS can have on the performance of a resisted isometric knee extension manoeuver by people with painful knees. A second goal was to determine the effect TENS can have on the subjective report of pain that occurs during the performance of an isometric test against a predetermined load. The study also examined the relationship between pain relief and muscle performance.

METHODOLOGY

Subjects The study subjects were selected from those persons with painful knees who were referred to the Williams Lake (British Columbia) Physiotherapy Clinic. Informed consent was obtained from each participant before testing began. The 12 subjects, 10 female and 2 male, ranged in age from 14 to 48 years (average age = 27). Of this group 10 presented with clinical evidence of

retropatellar pain while one complained of traumatic patellar tendonitis and another complained of knee pain following a recent tibial osteotomy. All patients complained of pain during resisted isometric knee extension. None exhibited any neurological deficit or muscle injury. Patients who presented with detectable joint effusions or who had recently been immobilized were excluded from the study.

Each subject received a true and placebo TENS treatment on consecutive days of testing. Those who received the true treatment on day 1 of testing received a placebo treatment on day 2 and vice versa. In this manner subjects served as their own control.

EQUIPMENT

True TENS The investigators in this study employed a Neuromod Selectra TENS unit (model 7750). During the treatment phase of the experiment, patients were managed with a conventional mode of TENS delivered to the affected knee for 30 minutes. Following a technique outlined by Mannheimer and Lampe (8), a current of 90 Hz with an 80 microsecond pulse width in a spike waveform was applied. A

dual channel criss-cross electrode placement technique was used (Fig 1). The goal of treatment was to provide a concentration of current flow at the patella so that electrical paraesthesia could be produced. Intensity was adjusted at regular intervals to ensure that the patient felt a firm but comfortable tingling sensation at the knee.

Placebo TENS During the placebo treatment phase of the study, subjects were informed that the goal of treatment would be to affect therapeutically, those nerves responsible for the transmission of knee pain as they enter the spinal cord. To this end, a set of electrodes were arranged bilaterally at the level of the third lumbar vertebrae. Stimulation parameters were similar to the true treatment regime; however, the intensity was set at a level only slightly above perceptual threshold and maintained there by periodic adjustment.

The Visual Analogue Pain Scale A visual analogue pain scale, (VAS) was used to measure each patient's subjective experience of pain during the tests.
The VAS consisted of a vertical line of 100 mm, with each end representative of the extremes of the sensation of pain. The upper extreme represented "pain as bad as it could be",

while the lower extreme represented "no pain at all". Each subject indicated the amount of pain experienced by a horizontal pen mark on the VAS. The point on the scale indicated by the subject at rest represented zero. The points marked at different times of pain assessment were measured from this point of reference in millimeters. A positive score was noted when pain increased and a negative score when pain decreased. Each time the sensation was to be recorded, a new scale was presented so that the patient could not base their momentary judgement on a preceding score.

Strength Measures All strength measures were made on a Series III-307 Unilateral Quadriceps/Hamstring Hydragym exercise device. Readings were in pounds force generated by the exercising subject. All strength measurements were taken with the patient performing an isometric contraction against a fixed lever arm adjusted to a position equivalent to 30 knee flexion. Once experimentation began no adjustments were made to the machine.

Procedure

Following administration of a VAS at rest, each

participant performed a maximal voluntary isometric knee extension manoeuver and the results were recorded. The subject then made a second effort to 80% of the maximal value, and another VAS test was immediately administered. The treatment phase of the study then began. Following a 30 minute true or placebo treatment a third VAS was administered. The subject then performed a post treatment maximal voluntary strength test and after a brief rest was asked to repeat the 80% maximal pre treatment strength manoeuver. A fourth VAS assessment was then completed.

Statistics

A 2 x 2 repeated measures analysis of variance was used to examine the effect of true and placebo TENS on the amount of force exerted by each subject. A similar analysis was used to determine the effect of TENS on the subjective report of pain experienced at 80% maximal voluntary work load. Results were tested by post hoc analysis, (Tukey "a", $p=0.05$). In an effort to investigate a possible relationship between pain relief and performance, correlational analysis was performed (Pearson Product Moment Correlation).

RESULTS

Analysis of the collected data revealed that following a true TENS treatment, patients significantly increased the amount of force they could exert ($p=0.009$). While in the true treatment phase of the experiment subjects demonstrated an average increase in force output of 11.23%. The placebo treatment was associated with no significant change in the force exerted (Fig. 2).

Regardless of the type of treatment administered TENS was associated with a significant reduction in the subjective report of pain experienced while performing an 80% maximal voluntary isometric contraction ($p=0.027$). In this respect the placebo was as effective as the true treatment technique (Fig. 3).

Correlational analysis determined that there was no relationship between pain relief and the amount of force exerted.

DISCUSSION

True TENS demonstrated a capacity to improve patient performance of an isometric strength manoeuver. In our results, subjects in the true TENS condition showed an 11.23% increase in force developed against a fixed lever arm. It is interesting to note that of the 12 subjects tested only seven were responsible for the improved scores recorded after a trial of true TENS. When these persons are considered alone the average increase in force is found to be 23.45%. This indicates that effective use of TENS may partly depend on the identification of those persons who respond favorably to this modality. Criteria for such a selection process have not been established.

This study found the placebo treatment to be as effective as the true treatment in reducing the subjective report of pain at a given isometric load. Despite this result the placebo treatment was not associated with any improvement in force output. In fact a slight deterioration in performance was observed after the placebo TENS. These results make it difficult to attribute the improved

performance following a true TENS treatment to pain reduction alone. In light of these findings other sources of inhibitory stimuli that may be altered by true TENS must be considered.

In a 1929 publication N.J. Blockey notes that pain alone does not exert the inhibitory influence credited to it (6). More recently Shakespeare and associates concur that pain is not the sole factor responsible for reflex inhibition (5). In several studies joint effusion has been demonstrated as a powerful stimulus for the production of reflex inhibition. Shakespeare et al identify the large diameter afferents as the pathway of input that results in reflex inhibition. They suggest that a gross joint effusion is not necessary to cause inhibition but synovial congestion or edema is as effective. Such a condition would supply the necessary stimuli to the large diameter afferents to cause the reflex to occur.

Using this model of inhibition it may be argued that the local application of TENS at sufficient intensity may partially block the inhibitory influence of the large diameter joint afferents. In this manner reflex inhibition is at least partially overcome. Until further evidence

becomes available the physiological explanation for the success of TENS will remain speculative.

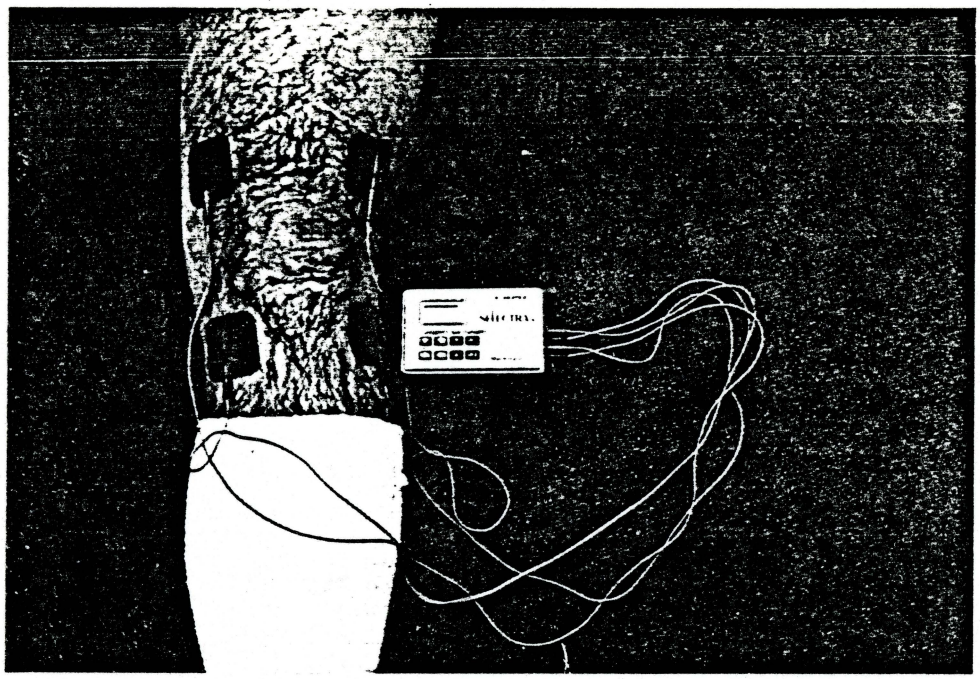
CONCLUSION

This study indicates that true TENS is effective in improving performance of maximal isometric knee extension manoeuvers among a group of patients with painful knees. The physiological explanation for this finding is unclear; however, this does not diminish the clinical significance of these results.

True and placebo TENS both reduce the subjective report of pain at a given joint load. There was no difference in the effectiveness of the two treatments in this regard. A correlation between the amount of pain relief and performance of a maximal isometric strength test could not be established.

TENS may be used effectively to augment the performance of individuals recovering from knee injury.

FIGURE 1: ELECTRODE PLACEMENT DURING A TRUE TENS TREATMENT



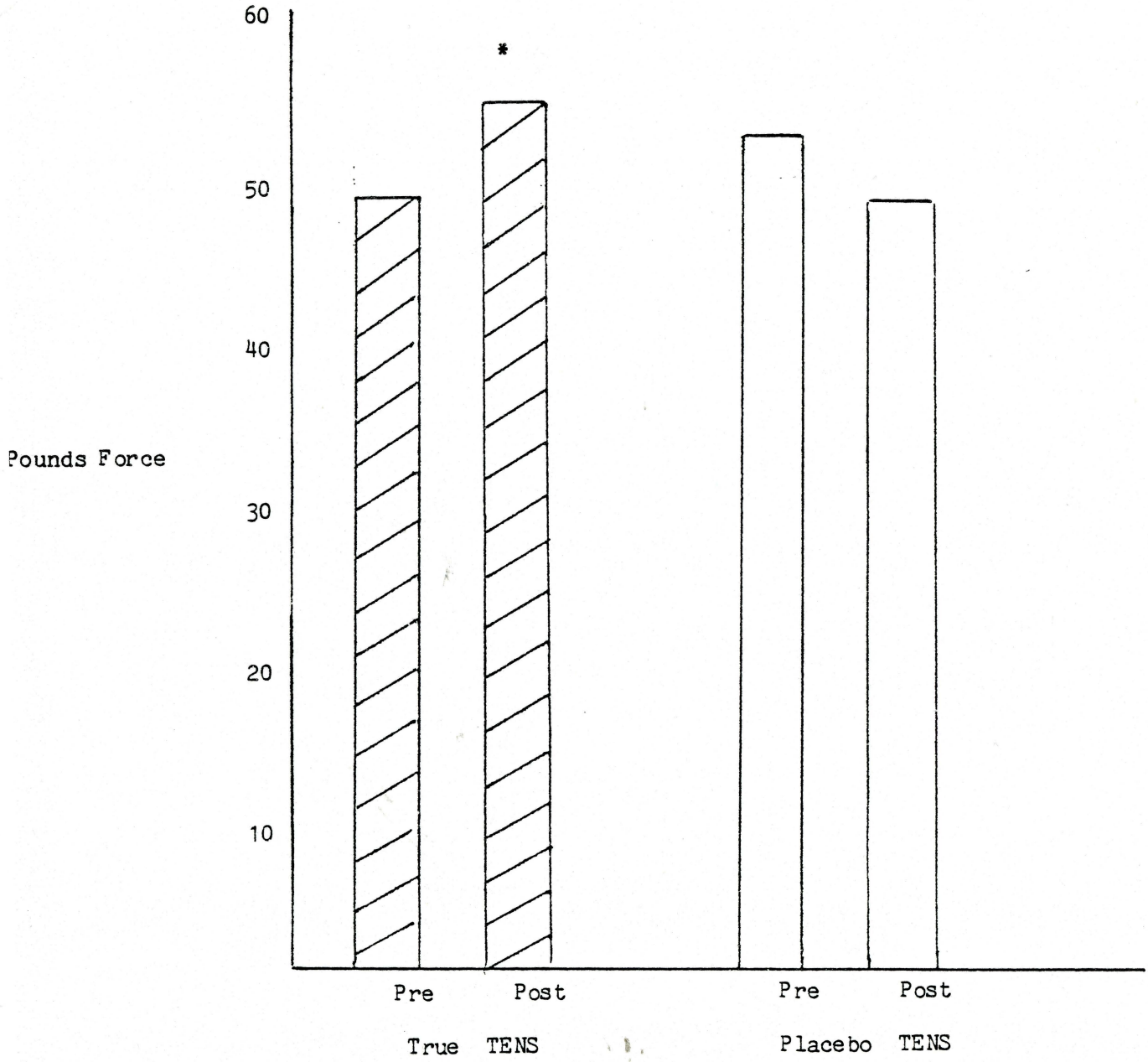
Electrode Placement

True TENS

	Channel A		Channel B
A_1	two inches above lateral aspect of patellar base	B_1	two inches above the medial aspect of the patellar base
A_2	below medial condyle of tibia at the level of the tibial tuberosity	B_2	Anterior and inferior to fibular head

FIGURE 2:

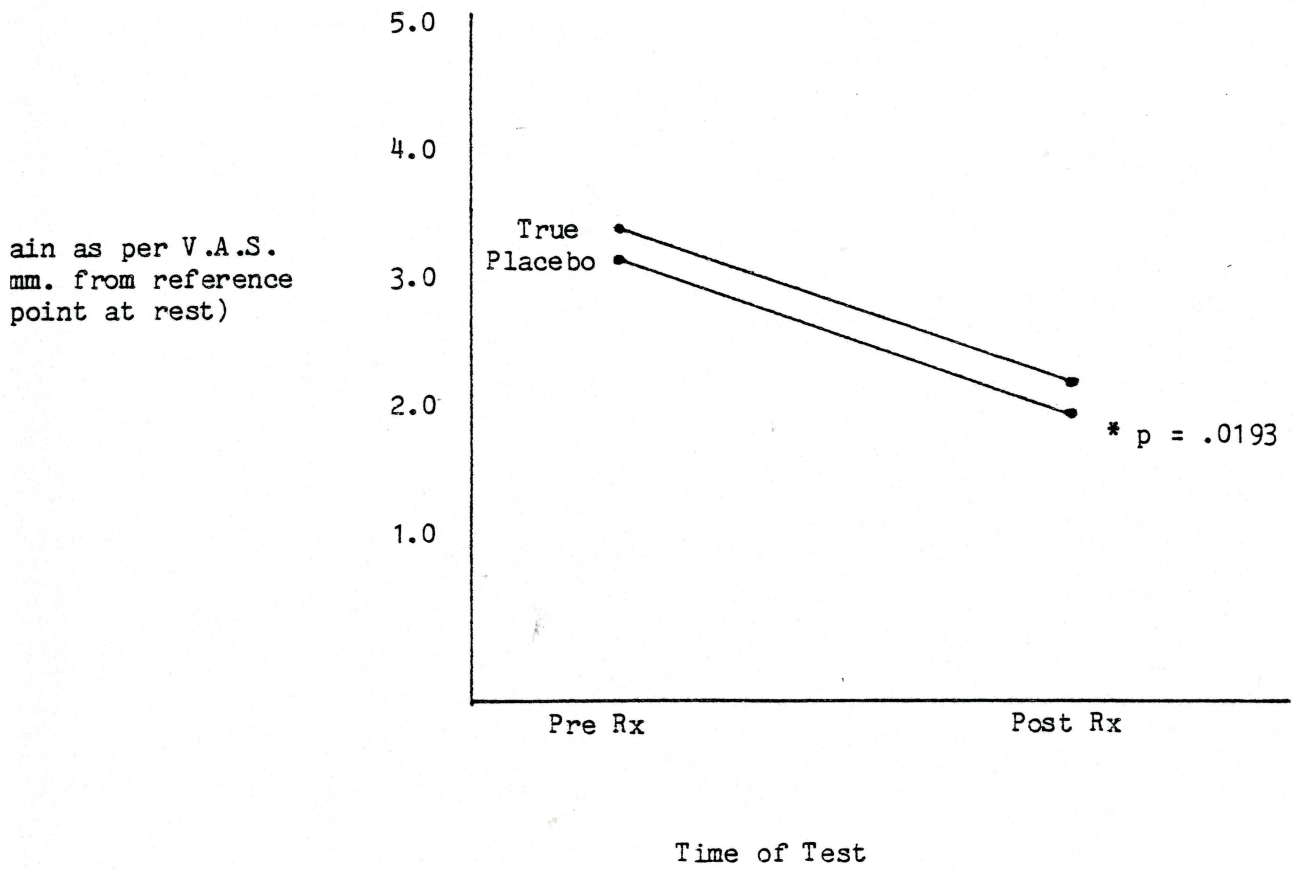
Force Generated Before
and After Treatment



* significant p = .05

FIGURE 3

Subjective Pain Experienced at 80% M.V.C.



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APPENDIX II

CALCULATION OF METHOD ERROR

II-A mean twitch tensions
II-B best MVC of 5 trials
II-C best % activation of 3 trials

SUBJECTS

Ten healthy subjects were tested from Jan. 25 to Feb. 5, 1986. Of this group 5 were male and 5 female. An equal number of tests were performed on the right and left lower extremities. Tests were carried out on 2 consecutive days and scores from day 1 to day 2 were compared. All measures were made with equipment used for data collection among the experimental populations.

STATISTICS

To determine the reproducibility of measures, the method error was calculated and expressed as a coefficient of variation (Thorstensson, 1976; Friman, 1977; Sale, 1982)

$$ME = \frac{\text{standard deviation of the mean difference}}{\text{the square root of two}}$$

$$V = \frac{ME}{(\bar{X}\text{-day 1} + \bar{X}\text{-day 2}) \div 2} \times 100$$

APPENDIX II-A
MEAN RESTING TWITCH TENSION (Nm)

($\bar{x}$ of 6 trials)

SUBJECTS	DAY-1	DAY-2	DIFFERENCE
1. E.F.	17.67	23.23	-5.56
2. H.C.	31.67	30.76	.91
3. J.Er.	9.58	12.25	-2.75
4. J.E.	20.74	26.32	-5.58
5. F.O.	45.07	37.44	7.63
6. M.C.	46.81	44.80	2.01
7. N.T.	51.25	50.72	.53
8. D.H.	39.09	37.74	1.35
9. L.B.	25.03	27.41	-2.38
10. A.B.	56.29	61.88	-5.59
	$\bar{x}$= 34.32	$\bar{x}$= 35.25	$\bar{x}$= -0.943
	SD= 15.74	SD= 14.47	SD= 4.25
	SE= 4.98	SE= 4.58	SE= 1.35

$$ME = \frac{4.25}{1.414} = 3.0 \qquad t = \frac{-0.943}{1.35} = 0.698 \quad (N.S.)$$

$$V = \frac{3.0}{(34.32 + 35.25) \div 2} \times 100 = 8.62\ \%$$

APPENDIX II-B
BEST MVC (N•m)

(best of 5 trials)

SUBJECTS	DAY-1	DAY-2	DIFFERENCE
1. E.F.	120.00	108.94	11.06
2. H.C.	197.54	201.68	- 4.14
3. J.Er.	48.24	58.81	-10.57
4. J.E.	167.38	162.40	4.98
5. F.O.	249.07	239.75	9.32
6. M.C.	181.98	163.29	18.69
7. N.T.	138.83	146.44	- 7.61
8. D.H.	250.02	238.17	11.85
9. L.B.	135.97	133.75	2.22
10. A.B.	194.08	194.47	- 0.39
	$\bar{x}$ = 168.31	$\bar{x}$ = 164.77	$\bar{x}$ = 3.54
	SD = 61.07	SD = 56.67	SD = 9.37
	SE = 19.32	SE = 17.93	SE = 2.96

$$ME = \frac{9.37}{1.414} = 6.62 \qquad t = \frac{3.54}{2.96} = 1.195 \quad (N.S.)$$

$$V = \frac{6.62}{(168.31 + 164.77) \div 2} \times 100 = 3.97\%$$

APPENDIX II-C
% ACTIVATION

(best of 3 trials)

SUBJECTS	DAY-1	DAY-2	DIFFERENCE
1. E.F.	84.80	93.98	- 9.18
2. H.C.	94.31	98.65	- 4.30
3. J.Er.	99.47	100 00	- .53
4. J.E.	93.30	100.00	- 6.70
5. F.O.	91.36	80.30	11.06
6. M.C.	85.31	89.91	- 4.60
7. N.T.	78.82	85.33	- 6.51
8. D.H.	83.27	81.25	2.02
9. L.B.	98.89	94.76	4.13
10. A.B.	99.13	91.16	7.97
	$\bar{x}$ = 90.86	$\bar{x}$ = 91.53	$\bar{x}$ = -.67
	SD = 7.41	SD = 7.33	SD = 6.78
	SE = 2.35	SE = 2.32	SE = 2.41

$$ME = \frac{6.78}{1.414} = 4.79\ \%$$

$$t = \frac{-.67}{2.14} = .31 \quad (\text{N.S.})$$

$$V = \frac{4.79}{(90.86 + 91.53) \div 2} = 5.25\ \%$$