"A controlled double blind study comparing the effects of strong Burst Mode, and High Rate Transcutaneous Electrical Nerve Stimulation, when both are applied to acupuncture points on osteoarthritic knees".

A study undertaken to complete the requirements of a Masters degree in Medical Science, in the Department of Community Medicine, Medical School, of the University of Tasmania, 1988.

by

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ABSTRACT

This study examined the effects of two different frequencies of Transcutaneous Electrical Nerve Stimulation (TENS) on chronically painful and stiff osteoarthritic knees. The effects studied were changes in pain state, changes in stiffness state, changes in circumference, and changes in range of movement.

Three groups, each of 20 subjects, were given a single 30 minute application of TENS: the first group received High Rate (Conventional) TENS, the second group received strong Burst Mode (Low Rate) TENS, and the third (control) group received a placebo application using the same active TENS with nonfunctioning leads. Each TENS frequency was applied for the same length of time, at tolerable intensities, to the same four acupuncture points around the knee.

Measurements of joint pain and stiffness using Absolute Visual Analogue Scales were made immediately before and after the TENS application. Objective measurements of joint circumference and range of movement were also made immediately before and after the test, using a tape measure and goniometer respectively. The length of time the post-test pain and stiffness relief lasted was determined by the subject reporting when his "normal" pain and stiffness returned. These reports were collected 24 hours after the test.

The aim of the experiment was to establish the hypothesis that strong Burst Mode TENS would produce significantly greater and longer lasting effects than those produced by High Rate TENS.

The results from the study did not entirely support the hypothesis, but the significance of the findings suggested that continuing investigation into TENS action is warranted.

This thesis contains no material which has been accepted for the award of any other degree in any University or College, and to the best of the candidate's knowledge and belief the thesis contains no material previously published or written by any other person except when due reference is made in the text of this work.

Karen Grimmer

HYPOTHESIS

The effects produced by the application of strong Burst Mode TENS will be greater and longer lasting than those produced by High Rate TENS. **

AIMS OF THE EXPERIMENT

1. This experiment aims to determine whether strong Burst Mode TENS produces significantly greater immediate pain and stiffness relief than that produced by High Rate TENS.

2. This experiment also aims to determine whether strong Burst Mode TENS produces significantly longer lasting pain and stiffness relief than that produced by High Rate TENS.

3. This experiment aims to determine whether strong Burst Mode TENS produces significantly greater immediate objective changes than those produced by High Rate TENS.

** The quantitative aspects of the hypothesis are covered in Section 6.2.

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CHAPTER 1.

INTRODUCTION

Arthritis.

The generic term "arthritis" refers to all the conditions that cause "stiffness, soreness and pain in joints, encompassing acute and chronic conditions" (Apley 1977). Shane & Grant (1987) suggest, after reviewing the 1983 statistics of the U.S.A. Health Department, that 80% of 40 million people over 55 years of age have some form of joint degeneration. Hadler (1985), in an analysis of U.S.A. workdays lost from arthritic related conditions, predicts that chronic arthritis affects at least 40% of the adult working population with varying degrees of incapacity. Chronic osteoarthritis exists in three main forms; osteoarthritis, rheumatoid arthritis and gout (Corrigan & Maitland 1986). Osteoarthritis, the subject of this study, is generally described as a "wear and tear" disease (Grote 1987), and it is the middle aged and elderly who are affected most. Although epidemiological studies have not established it, there appears to be a higher ratio of women suffering from ostearthritis than men; various studies suggest that it may be unequal and unknown (Maquet 1978), 2:1 (Hadler 1985) or 3:1 (Bland 1983). The preponderance of females in experimental studies by Taylor, Hallett & Flaherty (1981), Santiesteban (1983), Thorsteinsson, Stonnington, Stillwell & Elveback (1977,1978) and Mannheimer & Lampe (1984) seem to concur with a sex difference, although the reasons for the difference are not clear.

Osteoarthritis can be divided into two categories:

1. polyarticular degenerative arthritis, rarely occurring before 35 years of age,

2. monoarticular arthritis resulting from a particular joint surface incongruity occuring as a result of an injury or disease [Crenshaw (1971), Murray & Jacobson (1971)].

While the first category of osteoarthritis can affect joints indiscriminately, the weight bearing joints are mostly affected in the second category, as these are most likely to sustain injury. Of these, the hip and the knee are the most severely affected [Huskisson & Dudley-Hart (1978), Corrigan & Maitland (1986)]. It is considered that constant osteoarthritic pain would become chronic 6 months after diagnosis; after this time it could be expected that the joint pain would regularly affect the sufferer's lifestyle, mental attitude and physical expectations [IASP Subcommittee Report (1979), NH & MRC Report (1988)].

The knee joint has been chosen for this study because:

1. its affliction by chronic osteoarthritis is relatively common (McCarty 1979),

2. osteoarthritis of the knee is a separate and diagnosable condition using objective and functional measures (Cross & Crichton 1987),

3. the anatomy and function of the knee is well documented [Hollinshead (1969), Gray's Anatomy (1972), Brunnstrom (1979)],

4. the long term management of the pain and dysfunction in an osteoarthritic knee often is a problem for physiotherapist, doctor and patient (Smith, Lewith & Machin 1983),

5. despite TENS applications for osteoarthritic knees being described in previous experiments, the parameters for successful, replicable and lasting pain relief have not been adequately validated [Smith et al (1983), Taylor et al (1981), Mannheimer et al (1984)], and

6. the efficacy of pain relief with High Rate TENS and strong Burst Mode TENS applied to the same stimulation points at the knee has not been demonstrated.

Current treatment for osteoarthritis of the knee ranges from surgery, administration of analgaesic and anti-inflammatory drugs, heat and cold therapy, physiotherapeutic electrotherapy measures, to exercises and/or rest. The efficacy of drug therapy has been questioned (Taylor & Ghosh 1981), as the peak and valley effects and the systemic conditioning created by long term drug administration have been recognised as undesirable in the long term [Duncan (1982), Shane & Grant (1987)]. The efficacy of most physiotherapeutic treatments (such as Ultrasound, Interferential, Shortwave therapy and exercise) is largely untested with respect to osteoarthritic conditions [Santiesteban (1983), Smith et al (1983), Care, Harefield & Chamberlain (1981)], although physiotherapists have always believed in the personal contact value of these treatment methods (Partridge 1980). It has also been considered that diet may play a part in predisposing an individual to arthritic conditions, but this has not been rigorously scientifically tested [Pritikin (1985), Horne (1983), Phillips (1983)].

Transcutaneous Electrical Nerve Stimulation

The application of Transcutaneous Electrical Nerve Stimulation and its effects on osteoarthritic pain is the best documented of all currently available, non-invasive, electrotherapeutic, pain relief measures. Placebo studies have been conducted since the development of TENS first created medical interest [Long (1974), Thorsteinsson et al 1978)], and under controlled, blinded studies osteoarthritic conditions have been shown to respond well to Transcutaneous Electrical Nerve Stimulation [Mannheimer & Carlsson (1979) Dougherty (1979), Taylor et al (1981), Duncan (1982), Smith et al (1983)]. However, the optimal stimulation levels, electrode placements, lengths of stimulation time and pulse rates of appropriate TENS application are still disputed issues [Taylor et al (1981), Wolf, Gersh & Rao (1981), Medtronic (1982), Wolf & Gersh (1985), Mannheimer (1987)].

Although electricity has been believed to be a pain relieving tool since early Roman days (Stillings 1974), the Pain Gate Theory of 1965 created a resurgence of interest in its application for the relief of chronic pain [Melzack & Wall (1965), Shealy (1966)]. The Transcutaneous Electrical Nerve Stimulator was developed in the 1970's following awakened interest in a successful but forgotten model - the Electreat (Barcalow 1919). It was revised first in 1974 to become the Dorsal Column Stimulator, a screening device for potential dorsal horn implant patients [Shealy (1974), Long (1974)]. Such was the pre-surgery success of this unit as a pain relieving tool, that the Transcutaneous Electrical Nerve Stimulator was developed as a separate treatment entity (Hymes 1984).

Use of High Rate TENS (80 - 150 Hz), and more recently Low Rate TENS (2-3 Hz) has gained a place as an adjunct to medication and operative procedures to relieve chronic pain [Ersek (1977), Lampe & Mannheimer (1984)]. Strong Burst Mode TENS is an adaptation of Low Rate TENS, creating the same effects while using less intensity (Mannheimer 1987). The issue of which is the most effective TENS frequency in which circumstance is far from answered. Given that all treatment for chronic osteoarthritic pain has drawbacks, and that TENS therapy has been applied previously in clinical and experimental settings to osteoarthritic joints, this study aims to test whether strong Burst Mode TENS (pulse trains of 80 Hz internal frequency generated at 3Hz) produces significantly greater local pain and stiffness relief as well as significantly greater objective changes in osteoarthritic knees, when compared with High Rate TENS (80 Hz) and a placebo.

Pain Relieving Actions of Transcutaneous Electrical Nerve Stimulation

1. High Rate TENS

The initially accepted action of High Rate Transcutaneous Electrical Nerve Stimulation was the excitation of the larger, myelinated nerve fibres which immediately inhibit segmental nociceptor reception at the Substancia Gelatinosa in the Dorsal Horn of the Spinal Column [Burton & Maurer (1974), Ersek (1977)]. Consequently, it has been suggested that the action of High Rate TENS may be most effective during application, but that pain relief is unlikely to last longer than the treatment [Burton et al (1974), Ersek 1977)]. The original Pain Gate Theory has since been extrapolated to incorporate theories of central inhibitory balance and pattern specificity [Shealy (1973), Watson (1981b), Wall (1978)]. Recent studies indicating longer periods of pain relief, after High Rate TENS application, have been published, but with few definite reasons for the results [Smith et al (1983), Dougherty (1979), Mannheimer et al (1984), Taylor et al (1981)]. The inclusion of such concepts as pattern generation (Melzack & Loeser 1978), cognitive influence (Sternbach 1976) and higher cerebral input (Jacox 1977) into chronic pain reception mechanisms must be considered when discussing pain relief from High Rate TENS.

Whether High Rate TENS triggers intrinsic opiate release after longer stimulation times has been discussed by Lundberg (1984), and Andersson & Holmgren (1978), but equivocal answers have been compounded by varying experimental techniques and results [Hughes, Lichstein, Whitlock & Harker (1984), O'Brian, Rutan, Sanborn & Omer (1984)]. Given the relaying potential of the ascending and descending tracts of the Dorsal Horn of the Spinal Column, it is possible that along with its already expounded action at segmental spinal levels and the higher brain, High Rate TENS may trigger some intrinsic opiate production, thus promoting a pain suppression loop involving segmental and central processes (Bishop 1980).

2. Burst Mode and Low Rate TENS

The concept of Low Rate TENS was developed from acupuncture stimulation by Eriksson & Sjölund (1976). Burst Mode TENS is a recent, more comfortable adaptation of Low Rate TENS (Mannheimer 1987). Burst Mode TENS comprises trains of high frequency pulses generated at a low rate. At weak amplitudes, without muscle contraction, Burst Mode TENS can be used to produce a High Rate TENS effect, but at strong amplitudes, it has "the same net effect quantitatively and qualitatively as the single (Low Rate TENS) pulse at the same rate" (Mannheimer & Lampe 1984). It is this application which has been used in this study. Because of the analgaesic effect on patients with peripheral sensory neurological damage, low frequency TENS currents are thought to have little effect on the Pain Gate [Andersson & Holmgren (1975), Eriksson, Sjölund & Nielzen (1979)]. Low Rate and strong Burst Mode TENS produce a pulsed local afferent/efferent action which is too slow and generalised a stimulus to selectively conduct via fast acting myelinated fibres [Andersson (1978), Andersson, Hansson, Holmgren & Renberg (1976)]. Explanations of Low rate TENS action have been enhanced by the discovery of its effect on the intrinsic opiate release mechanisms in the spinal cord and brainstem [Synder & Matthyss (1975), Hughes, Smith, Kosterlitz, Fothergill, Morgan & Morris (1975), Eriksson et al (1979)]. Strong Burst Mode TENS has been shown to produce the same intrinsic opiate effect [Eriksson & Sjölund (1976), Sjölund, Terenius & Eriksson (1977)].

Low Rate (and strong Burst Mode TENS) produce rhythmic muscle contractions. This action is considered essential for the stimulation of the intrinsic opiate mechanisms: the stronger the contraction, the more effective the intrinsic opiate stimulation (Melzack & Wall 1984). Low Rate TENS current must be applied at uncomfortable intensities for 30 minute or more (Holmgren 1975) to stimulate the release of intrinsic opiates (particularly B-endorphin) from central brainstem areas. A negative feedback loop of pain suppression is thus possibly effected (Watson 1982). Low Rate TENS currents have a slow but increasingly powerful action during stimulation (Andersson 1979). It is known that intrinsic opiates affect peripheral synapses as well as synapses centrally, and investigation into Low Rate TENS action on peripheral synapses is under review (Mannheimer 1987). Until the development of Burst Mode TENS, difficulty in establishing a comfortable effective stimulation intensity restricted the use of Low rate TENS as a pain relieving modality (Melzack & Wall 1984). Strong Burst Mode TENS requires less current than Low Rate TENS to produce a strong muscle contraction, and thus may be better tolerated by the patient in pain (Eriksson, Sjölund & Nielzen 1979).

Mannheimer (1987) considers that Low Rate TENS creates longer lasting pain relief (2 - 6 hours) than High Rate TENS because of the continuing action of the released intrinsic opiates. The literature promoting its use for orofacial pain (the best documented use, to date, of Low Rate TENS) suggests that it may create relief from chronic pain up to 18 hours after application [Andersson & Holmgren (1975), Chapman, Wilson & Gehrig (1976), Chapman, Chen & Bonica (1977)]. Andersson (1979) suggests that the muscular activity produced by Low Rate TENS increases the local blood supply, decreases local swelling and improves the surrounding muscle activity. Mannheimer (1987), and Lampe & Mannheimer (1984) indicate that strong Burst Mode TENS achieves the same effect. Given the need to avoid increasing the discomfort of subjects in the study, Burst Mode TENS of 3Hz pulse train with an internal frequency of 80 Hz was used to create the effects of a Low frequency TENS current.

Application of TENS to Osteoarthritic Knees

Consistently successful applications of TENS are slow to be established for specific conditions. Different, although apparently successful, applications of TENS to osteoarthritic knees are suggested by Mannheimer et al (1984), Thurin, Meehan & Gilbert (1980), Dougherty (1979), Andersson et al (1976), Melzack (1976), Smith et al (1983), Taylor et al (1981) amd Melzack et al (1984). Variations in parameters used in these trials highlight the dilemma of researching this field. The manufacture of increasingly sophisticated TENS apparatus is outstripping clinical knowledge of appropriate use of TENS options. Lundberg (1984), Mannheimer & Lampe (1984) and Wolf & Gersh (1985) urge continued, controlled clinical experimentation. Inadequate data, poor experimentation techniques, inadequate controls and confounding factors have plagued research in TENS use for pain control. Symptom relief by TENS for sufferers from osteoarthritis of the knee would improve daily lifestyle, and would reduce lost work time, use of medication, visits to medical personnel and surgery waiting lists.

Use of Acupuncture Points for Electrode Placements

Although the clinical use of acupuncture points for electrode placements has been suggested for both High Rate and Low Rate TENS, the reasons for the choice are different. The use of acupuncture points for High Rate TENS application has been mainly for convenience [Thurin et al (1980), Mannheimer et al (1984), Smith et al (1983)], because the action of High Rate TENS is considered to be neural blocking. As Low Rate TENS is an adaptation of acupuncture, the placement of Low Rate TENS electrodes has always been over acupuncture points to enhance maximum endorphin stimulation [Andersson, Eriksson & Holmgren (1973), Andersson & Holmgren (1975), Melzack (1976), Melzack, Stillwell & Fox (1977)]. Use of acupuncture points is considered necessary for successful applications of Low Rate TENS (Mann, Bowsher, Mumford, Lipton & Miles 1973). However, the size of the TENS electrodes used in all these studies would make the stimulation of the pure acupuncture point questionable. Indeed, the same comment could be made about this study. The size of TENS electrodes presently available would make the isolated stimulation of acupuncture points impossible. Acupuncture points can be a guide only to anatomical placement.

Acupuncture points on the medial (Spleen 9), lateral (Gall Bladder 33), posterior (Urinary Bladder 40) and anterior (Spleen 10) aspects of the knee have been used for this study. By stimulating over these points, most of the cutaneous nerves of the knee would be involved in the treatment, the entire knee joint would be irradiated (Essentials of Chinese Acupuncture 1980), and the four most painful areas of the osteoarthritic knee suggested by Smith et al (1983) would be covered.

The Placebo

The use of a nonfunctioning TENS to authenticate clinical experimentation has been discussed by Smith et al (1983), Hansson & Ekblom (1983), and Thorsteinsson et al (1978). Given the guidelines laid down in NH and MRC Statement on Human Experimentation and Supplementary Notes (1983), it is suggested that such placebo application constitutes a valid form of clinical experimentation. However, it has been suggested that a placebo application may itself induce the production of endomorphins [Levine, Gordon & Fields (1978), Skrabanek (1978), Chen (1980)], and it is possible that a placebo application of TENS may not give the baseline levels of response to TENS that are expected. Due to the inadequacy of most longterm medication and physiotherapy regimes for ongoing relief for osteoarthritis of the knee, the mere possibility of symptom reduction may create expectation of a result.

Measurements

1. Subjective

The Absolute Visual Analogue Scale, chosen for this experiment, is considered an appropriate method by which clinical pain can be recorded (Zussman 1986). The inherent intervals are considered equal (Stewart 1977), the accuracy can be calculated to one decimal point (Scudds 1983) and the 10 cm continuum is considered statistically viable (Revill, Robinson, Rosen & Hogg 1976). The patient is not limited by lack of choice of descriptive words (Price, McGrath, Rafii & Buckingham 1983), nor by having to read and understand English (Stewart 1977), although the abstract quality of the scale "may frustrate some subjects" (Kremer, Atkinson & Ignelzi 1981). Zussman (1986) considers the scale "a reliable, convenient, inexpensive and readily analysable method of measuring subjective intensity".

The measurement of stiffness by the Absolute Visual Analogue Scale has no precedent in the literature reviewed for this study. Stiffness is inherently a subjective measure, although change in stiffness is possibly related more to objective changes in the joint. Both Melzack (1975) and Stewart (1977) considered the Absolue Visual Analogue Scale a successful measuring device for any subjective assessment. Thus for ease of administering this experiment, a second, parallel, Absolute Visual Analogue Scale was used to measure immediate change in stiffness.

Verbal reports of length of pain and stiffness relief have been reported previously [(Smith et al (1983), Taylor et al (1981), Dougherty (1979), Mannheimer et al (1984)]. This author accepts the possibility of inaccurate reporting. However, it has been suggested by Altman (1986) that chronic sufferers of osteoarthritis of the knee are capable of being accurate when recording the diurnal variations of their pain, as they learn over time to accommodate their daily activities to their pain state. In this trial, subjects were asked to indicate when, over the 24 hour period following their TENS test, their post-test pain and stiffness returned to "normal".

2. Objective

The use of a goniometer to measure changes in knee joint range is discussed by Gifford (1914), Moore (1949), Twomey (1978) and Dougherty (1979). It is a commonly used tool in physiotherapy practice and provides an accurate measure of pivotal joint performance. Likewise the use of a tapemeasure to measure changes in joint circumference has been well established [Jacox (1977), Corrigan & Maitland (1986)]. Objective assessment is considered to be free of the bias that may affect subjective pain assessment [Berry & Huskisson (1972), Merskey (1974)], and it is felt that the inclusion of objective quantative measures may validate any improvement in knee joint function that this TENS application may produce.

Conclusion

TENS therapy is a possible adjunct to all forms of current treatment for osteoarthritis of the knee. It is claimed to be safe, painless, nonaddictive and easy to use [Ersek (1977), Hymes (1984)], and has application in both home and clinical settings (Mannheimer 1987). Its use has been dismissed as ineffective and painful (Griffin & McClure (1981) although it is possible that the method of application was inappropriate. Copious literature appeared in the late 1970's, exploring the use of TENS for a multitude of conditions. Poor experimentation technique, lack of specificity of conditions, and incomplete understanding of the biochemical actions of TENS caused many of these studies to be dismissed despite the invitation inherent in them to continue the research with more refined techniques. However, since the early 1980's, there has been little more written on TENS therapy, despite the continuing lack of successful specific TENS parameters for particular conditions. The availability of increasingly sophisticated Transcutaneous Electrical Nerve Stimulators without the experimental literature to match, has highlighted the need for continuing research into the finer points of TENS application. It is hoped that this experiment will clarify the frequency variable of TENS for chronic osteoarthritic pain relief.

Summary of the Study.

This double blind, randomised, controlled study compares the effects produced by High Rate and strong Burst Mode (Low Rate) TENS, when both are applied for the same time, at tolerable intensities, at acupuncture points on painful osteoarthritic knees. Three groups of male and female subjects with present osteoarthritic knee pain were tested with one application of either High Rate TENS, strong Burst Mode (Low Rate) TENS or a placebo. Measurements of pain, stiffness, circumference and range of movement were taken before and after the test. This study, using a specified protocol for TENS administration, aimed to determine the most successful frequency of TENS for the relief of symptoms of osteoarthritis of the knee. Possible confounders such as the season, test time, and medication were controlled as far as was possible. The subject's sex and pain threshold were addressed by randomly assigning the subjects into the three test groups.

Confounders which may have influenced the results were the subject's age, his expectations, his diet, and the length of time the subject had suffered from osteoarthritis. All measurements were taken by one person, who was independent of the study design and unaware of which TENS frequency was being administered to which subject. Because of its endorphin action, and the expected local tissue changes resulting from its effect on muscle contraction, it was hypothesised that strong Burst Mode TENS would produce significantly greater and longer lasting effects on chronic osteoarthritic knees than would High Rate TENS or the placebo. However, at 95% significance, it was found that strong Burst Mode TENS was not always the only effective TENS frequency.

Key Findings

1. Strong Burst Mode TENS, High Rate TENS, and the placebo all produced significant pain and stiffness relief, when comparing pre- and post-test Absolute Visual Analogue Scale readings.
2. Strong Burst Mode TENS produced a significant length of post-test pain relief only when compared with the placebo.
3. Strong Burst Mode TENS did not produce significant immediate post-test stiffness relief when compared with High Rate TENS and placebo.
4. Strong Burst Mode TENS and High Rate TENS both produced significant lengths of post-test stiffness relief when compared with the placebo.
5. Strong Burst Mode TENS produced significant immediate post-test change in joint circumference only when compared with High Rate TENS.
6. Strong Burst Mode TENS produced significant immediate post-test change in range of movement only when compared with the placebo.

The lengths of pain and stiffness relief produced in this study, indicate that the actions of High Rate TENS and strong Burst Mode TENS could support further investigation. The choice of Transcutaneous Electrical Nerve Stimulator, the length of the TENS application, the subjects' age, the length of osteoarthritis diagnosis, the use of acupuncture points for electrode placement and the study design may have mediated pain and stiffness relief beyond that which was anticipated.

OSTEOARTHRITIS AND THE KNEE

2.1 The Disease of Osteoarthritis

In the literature reviewed for this work, this author found no generally accepted standard criteria for the diagnosis of osteoarthritis [Darby (1983), Hadler (1982), Bland (1983), Sokoloff (1969)]. Clinical criteria for the diagnosis of this disease are currently being developed by the American Rheumatism Foundation (Altman 1986). Clinical findings often differ from joint to joint, but common to all subjects with osteoarthritis are morning stiffness, changes in radiological joint composition, and the average age of sufferers being over forty (Altman 1986). Physiotherapists generally rely on a diagnosis of osteoarthritis to be made by the referring doctor. Lack of a clinical diagnosis necessitates nonspecific treatment. This is not cost or time effective.

As the result of a study of physiotherapy attendance hours at 150 Community Clinics in Michigan, U.S., in 1984, Stross, Banwell, Wolf & Becker (1986) suggest that radiologically confirmed osteoarthritis is one of the most common and debilitating diseases prevalent in modern society. It is characterised by "defects in articular cartilage and related change in subchondral bone, joint margins, synovium and para-articular structures" (Altman 1986). Osteoarthritis has been documented since the days of Hippocrates (Darby 1983), although it was not until the 18th century that it was described separately from gout, and was recognised as a non-inflammatory and degenerative condition (Maquet 1976). In addition to the deterioration and abrasion of articular cartilage in affected joints, Sokoloff (1969) notes that osteoarthritis is recognisable "by formation of new bone at the articular surface".

More women than men are affected by osteoarthritis (Corrigan & Maitland 1986) although the reasons for this are unclear. Studies by Hadler (1982), Taylor, Hallett and Flaherty (1981) and Bland (1983) indicate that women may be affected in the order of 2 or 3:1. As osteoarthritis usually affects people over forty, menopausal interference in joint function, suggested by Maquet (1976), may indeed be an issue. It is possible that changes in leg angles and heel strike caused by womens' fashionable footwear (high heeled shoes) and abnormal thigh action (obesity) may cause a higher incidence of osteoarthritis. Increased female pelvis/ femoral angle (Kendall, Kendall & Boynton 1952) may alter angular loading on weightbearing joints (particularly the knee) in women, thus predisposing it to degenerative changes.

2.1.2 The Causes of Osteoarthritis

The causes of osteoarthritis are well documented. Cameron & Mehta (1984) suggest that osteoarthritis develops as a result of overloading the joint: by obesity, joint surface incongruity, or from normal loading on an abnormal joint consequent to trauma or disease. Darby (1983) and Altman (1986) mention the traditional separation of primary, or ideopathic, from secondary forms of osteoarthritis. Such secondary causes may be post traumatic, or as a result of congenital or developmental diseases, bone and joint disorders such as septic arthritis and osteoporosis, mechanical disorders like unequal limb length, or metabolic, endocrinal or neuropathic disturbances.

Analytical studies [(Maquet (1976), Radin (1973)] suggest that osteoarthritis is not caused by any single factor and that research to establish "a unique causative mechanism" will not succeed. Murray & Jacobson (1971) report radiological studies of a large proportion of ideopathic osteoarthritics as having pre-existing abnormalities, while Solomon (1976) found that an identifiable cause of the disease could be isolated in 92% patients studied with degenerative joint disease. Perhaps the separation of ideopathic from secondary causes may not be as clear as was thought by earlier researchers.

Some dispute exists however as to the exact trigger of the disease onset. Darby (1983) quotes variations in pathological presentation of the disease at autopsy, that expose the underlying lack of understanding of its mechanisms; "Why does fibrillation in some parts of the joint surface lead to osteoarthritis, while in others it is of limited progression?.... Is the synovial membrane an innocent bystander or does it play a leading role in initiating the disease process?" Altman (1986) notes that aspirated fluid from the synovium of an acutely inflammed osteoarthritic peripheral joint shows increased viscosity, with an increased white blood cell count, and raised protein and glucose levels. He quotes operative findings in that the cartilage of an affected joint also differs from that of a healthy joint, being frayed, ulcerated, yellowed and of irregular depth.

The illustration below looks at factors that may contribute to the establishment of osteoarthritis in a joint: "Clinical osteoarthritis appears to be the end result of a complex series of interconnected vicious circles.... one or more entry points into the series seems possible" (Darby (1983).

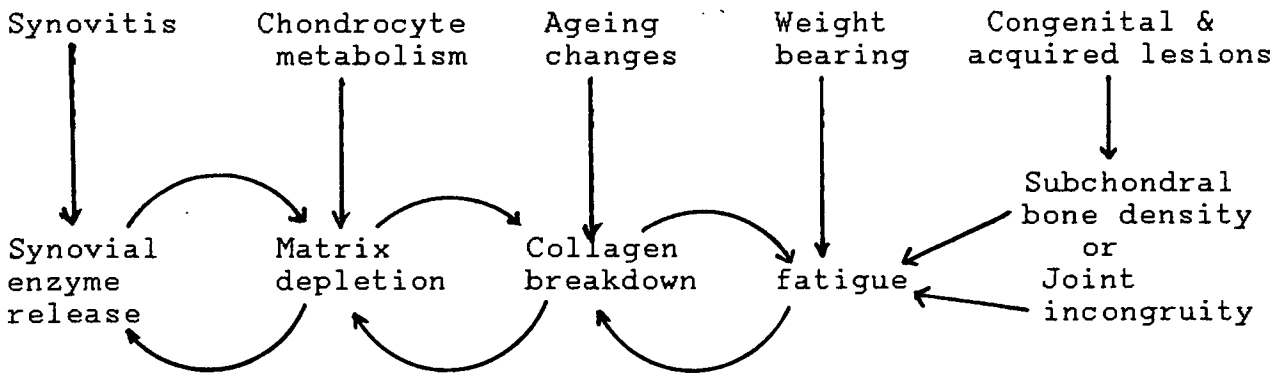


FIG. 2A Factors contributing to the establishment of osteoarthritis

2.2 Osteoarthritis of the Knee

The knee is one of the most common sites in the body for the establishment of osteoarthritis, as it is directly affected by injury, abnormal weight bearing patterns, obesity and joint incongruity (Adams 1971). Osteoarthritis can occur unilaterally in the medial compartment, the lateral compartment and/or the patellofemoral compartment. It can also occur in a generalised form throughout the joint. The medial compartment is the most common site of the disease, as it is constantly under load by postural alignment and body sway (Cameron & Mehta 1984).

Osteoarthritis of the knee may be caused by several precipitating factors. It is accepted that the presence of the meniscus plays an important role in maintaining joint surface congruity (Gray's Anatomy 1972) and changes in its state, or its absence, may alter the forces experienced across the joint during normal weight bearing, thus predisposing the joint to osteoarthritic changes (Maquet 1976). Although obesity is often quoted as a cause of osteoarthritis of the knee, both Sasaki & Yasuda (1987) and Altman (1986) suggest that it may be the altered heel strike and subsequent varus knee and heel posture that occurs as a result of obese thighs that may precipitate constant wearing of the knee joint surface. Joint incongruity or altered leg length from previous bony trauma will also alter weight bearing posture and thus encourage abnormal joint wear.

On researching this paper, the most recent, comprehensive list of clinical signs of osteoarthritis of the knee appears in Altman (1986);

" 1. Pain; on weight bearing, aching, present on most days.
2. Age; usually over 40 years.
3. Morning stiffness for >30 minutes.
4. Crepitus on active movement.
5. Bony joint margins; tender, palpably enlarged.
6. Palpable effusion; relatively cool or none.
7. Synovial fluid; clear, viscous, contains < 2,000 WBC/ml.
8. Changes in bony conformation as seen on X-ray."

2.3 Symptoms of Osteoarthritis relating to the Knee.

The symptoms of osteoarthritis of the knee combine subjective reports of pain and lack of function, objective assessment of changes in joint shape and strength, and radiological definition of joint destruction [Calabro (1986), Altman (1986)].

2.3.1 Pain

Apley (1977) writes: "Pain is the major symptom of O.A. In early stages it is present on waking, and then wears off with movement. With further use, the joint begins to ache. As the disease progresses, pain increases in severity and consistency, sometimes disturbing sleep." An observational study by Helal (1965) divides the pain of primary osteoarthritis into muscular, venous and capsular origins. With respect to the knee:

1. Muscular pain occurs in the quadriceps, hamstring and gastrocnemius muscles. It is cramplike on activity and improves with rest. It occurs as a result of muscle fatigue, and metabolic waste accumulation, in muscles which have been allowed to weaken through disuse.

2. Venous pain is "a dull aching or throbbing ....felt around the knee, usually worse towards the end of the day and persisting for a while after retiring to bed" (Helal 1965). It may be as a result of deformation of bone sinuses, thus pooling blood in the joint endings. This pain is thought to involve C fibre activity (Mackenny & Harris 1983).

3. Capsular pain is sharp pain felt on joint movement. It is the most common feature of osteoarthritis of the knee (Apley 1977), and is a result of capsular contractures being stretched during knee movement. Cyriax (1977) notes the pain felt at the end of range of movement may be capsular stretch rather than muscle spasm as was thought previously, and thus because of its acute, protective nature may involve A delta fibres (Helal 1965).

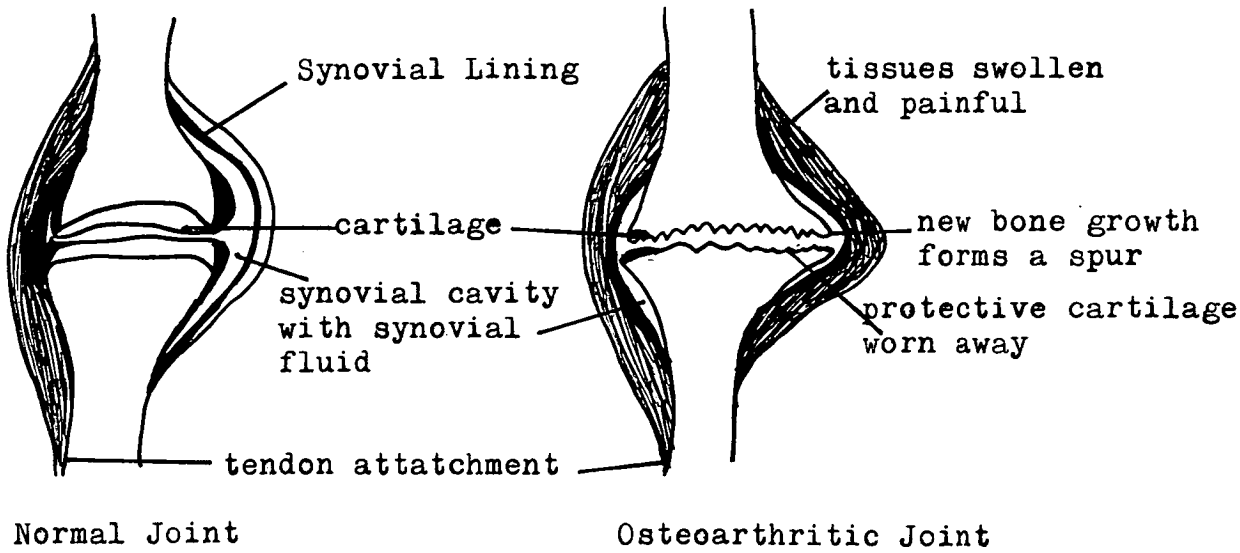
   It has been suggested that the capsular and ligamentous structures of joints are also richly supplied with C fibres (Mannheimer, Lund & Carlsson 1978), which may account for the diffuse ache which persists deep in the joint despite conservative medical attention (Corrigan 1986).

4. Cyriax (1977) describes another pain type, that of "grinding bone on bone when articular cartilage is worn away." This is considered mostly A delta pain transmission as cartilage has no nerve supply, while periosteum is well supplied with faster acting afferent nerves (Apley 1977).

   Melzack and Taenzer (1977) spoke of pattern generating mechanisms in the dorsal horn of the spinal column. It is possible that chronic osteoarthritis results in abnormal pain patterns being generated, thus establishing avoidance behavior (Merskey & Spear (1967). Osteoarthritis sufferers are aware of their limitations and may impose restrictions on their daily activities in the hope of minimising their pain (Altman 1986). The diurnal pain variation, functional changes with respect to the weather, and the effect of some foods, activities, and even wearing apparel have been noted [Jacox (1977), Berry & Huskisson (1972), Clark & Spear (1964)].

2.3.2 Joint shape and strength (FIG 2B)

Swelling and deformity are evident in most chronic osteoarthritic joints. The knee is more prone to OA involvement (Mackenny & Harris 1983) because of the complicated mechanics involved in knee movement, and because of its propensity for attracting trauma. Maquet (1976) noted that forces "up to three times the body weight cross the knee joint during normal walking", which lead Mackenny et al (1983) to suggest "that minor derangements in function may produce excessive localised abnormal loading leading to cartilage failure".



Scale: Approx. I in IO

FIG. 2B Differences between normal and osteoarthritic joint

Orthopaedic factors such as quadriceps weakness and genu valgum/ varus, and forefoot/rearfoot instability, may result in "unicompartmental degeneration of cartilage" given normal useage over time (Crenshaw 1971). Such quadriceps weakness will be apparent as wasting, and the underlying joint may appear more noticeably disfigured (Apley 1977). Soft tissue changes such as inflammed &/or thickened synovium may be objectively noticeable, as will swelling infiltrating the infra- and suprapatella bursae (Darby 1983).

Joint instability may be detected, as weakness resulting from frayed cruciate, medial and/or lateral collateral ligaments may not be overridden by the disease-mediated fibrosis of the ligamentous and capsular interconnections. Destruction of the articular cartilage and the menisci may also predispose to instability, especially if the quadriceps and hamstring static balance is not maintained [Corrigan and Maitland (1986), McConnell 1986)].

2.3.3 Radiological evidence

The most commonly used method of diagnosing osteoarthritis of the knee is by X-ray. Features recognised when diagnosing osteoarthritis include;

1. Joint remodelling. (FIG. 2C)

This is the most striking feature of osteoarthritis of a joint. Unfortunately confirmed by autopsy findings, it indicates that the disease process is well established (Mackenny et al 1983). Flattening of the weight bearing area of the bones involved occurs because of the destruction of cartilage, and the change is made more apparent by the formation of osteophytes at the margins of the joint, thus expanding the periphery of the joint profile. "Regressive remodelling" occurs centrally in the joint by way of osteoblastic activity, following surface destruction. "Progressive remodelling" occurs at the joint edges when articular cartilage reverts to its original childhood function of a growth plate (Darby 1983).

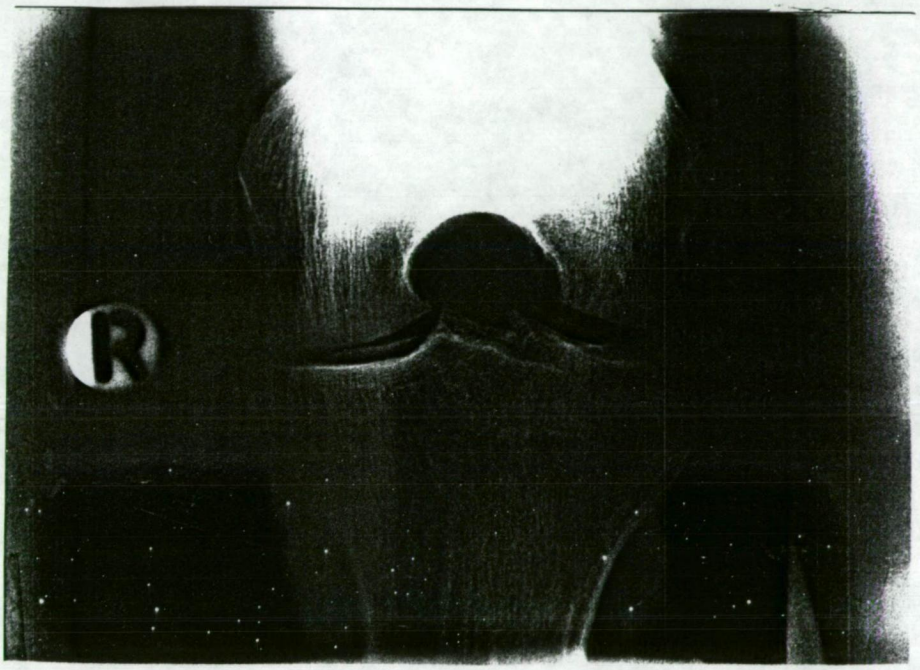


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FIG. 2C X-ray of osteoarthritic knee

2. Pathological changes in cartilage.

Anatomical studies lead Crenshaw (1971) and Cyriax (1977) to agree that degeneration of articular cartilage is "the hallmark, if not the actual cause, of osteoarthritis". The initial change appears as fibrillation of the superficial layers of the cartilage, involving disruption of collagen fibres in the mattrix. Clefts occur in progressive degeneration of cartilage "extending vertically and/or tangentially" (Darby 1983) into the deeper cartilage layers, leading to eventual fragmentation of the superficial cartilage. Abrasive action from the opposing joint surface increases the loss of cartilage once the destructive mechanism is established. Increase in the activity of articular chondrocytes can be seen adjacent to the clefts, as a result of cellular proliferation - a protective reaction to the inherent destruction of cells. This reaction can be observed in pathology tests, and when well established, in X-rays. (Cyriax 1977).

3. Changes in Subchondral Bone.

As destructive changes occur in superficial cartilage, the underlying bone is exposed to the erosive activity of the opposing joint surface. This leads to loss of end-bone substance, and a polishing effect known as "eburnation" occurs (Apley 1977). Friction and direct trauma lead to the loss of osteocytes, which may present as local avascular necrosis of bone, or fractures of the subchondral bone in advanced cases. However, the surviving bone cells show an increase in activity; weightbearing bony areas show higher than normal levels of osteoblast and osteoclast activity, resulting in radiological evidence of sclerosis (Crenshaw 1971).

4. Subchondral pseudo- cysts

This feature is found in other pathologies besides osteoarthritis, and the description is a radiological rather than a histological one. Cysts are areas of bone replaced by fibrous tissue, sometimes containing viscous fluid, and the whole area is usually lined with sclerotic bone. It is thought (Darby 1983) that the cysts result from high intra - articular pressure, which forces synovial fluid into the cavities in articular cartilage and subchondral bone created by earlier stages in the disease. However, because pseudo - cysts are also demonstrated in other pathologies whose etiologies differ from osteoarthritis, the real reason for their formation is not known.

2.4 Treatment of osteoarthritis of the knee

The objectives of all treatment of osteoarthritis of the knee are to reduce pain and to improve function [(Altman (1986), Calabro (1986), Stross, Banwell, Wolf & Becker (1986)].

Common methods can include one or more of the following:

1. surgery to reduce the loading on the knee or to replace the joint
2. physiotherapy procedures to relieve local pain and to increase strength in the surrounding protective muscle groups,
3. altering the angle of heel strike by shoe inserts,
4. pharmaceutical agents and
5. joint protection by external aids.

2.4.1 Surgery is employed when the destruction of the joint defies conservative treatment. It commonly takes two forms; tibial osteotomy to relieve the load on the joint, or total knee relacement (Corrigan & Maitland 1986). Joint debridement and unicompartmental arthroplasy are mentioned for younger sufferers (Cameron & Mehta 1986). The improving nature of artificial joint materials means that the age of total knee replacement patients is gradually lowering, although it would appear that surgical intervention is still a last resort (Sasaki & Yasuda 1987).

2.4.2 Physiotherapeutic procedures used in the conservative management of osteoarthritic knee pain vary. A critical issue is that so many of the electrotherapy measures used are untested with respect to their actions on osteoarthritis (Santiesteban 1983). Heat, ice and massage are often used in the management of acute joint pain and muscle spasm (Altman 1986), while ultrasound, Diathermy, Interferential, Laser therapy, and Transcutaneous Electrical Nerve Stimulation are suggested

for increasing the blood supply, decreasing sensory nerve irritability and improving muscle function [Taylor et al (1981), Smith, Lewith & Machin (1983), Scott 1969)]. Regular exercise to improve muscle performance, and strength and balance around the knee are vitally important to protect joint movement and compensate for absent or weak ligaments [Cameron & Mehta (1986), Care, Harefield & Chamberlain (1981)]. Assessment of posture, advice on the use of shoe raises or wedges and the important task of educating patients in lifestyle and use of aids to relieve joint load is also a physiotherapist's charter (Altman 1986). Partridge (1980) notes that empathy and concern in the overall care of chronic arthritics is not to be overlooked in treatment value.

2.4.3 Pharmacological agents are usually involved in successful conservative management of osteoarthritis of the knee. It was important that medication be recognised as a possible confounding factor in this trial, and thus be controlled in the study design.

Four classes of drugs are commonly used.

1. Anti-inflammatory agents including aspirin and some non-narcotic, nonsteroidal drugs are given to reduce inflammation at the joint site. These drugs are antipyretic, and are specific in blocking the actions of irritating chemicals on the cell membrane. Unfortunately, adverse multisystem reactions reported by some patients for all of the current NSAIDs on the market suggests that prescription of long term antiinflammatory drugs alone may not be indicated [Calabro (1986), Shane & Grant (1986)].
2. Muscle relaxants or antispasmodic drugs have a place in inducing relaxation in the soft tissues surrounding the affected joint.
3. Analgaesics for short term pain relief are an important measure in combatting acute exacerbations of osteoarthritis, but undesirable peak and valley effects of regular medication, and the systemic conditioning problems associated with long term drug administration are noted by Taylor & Ghosh (1981) and Duncan (1982).
4. Depocorticosteroids are valuable for an inflammed joint, but are not desirable for longterm management (Altman 1986).

2.4.4 Aids useful in the management of osteoarthritis of the knee range from walking sticks, canes and knee braces to pickup sticks, altered seat heights, and ramps to reduce joint load. If the patient is overweight, a diet may be indicated. A change in diet to reduce fats may also be suggested, as it is indicated by Grote (1987), Bland (1983), Pritikin (1985) and Horne (1983) that an improvement in osteoarthritic joint function can occur if dietary intake is higher in fibre and lower in fats.

2.5 Conclusion

Osteoarthritis is a debilitating disease which affects not only the individual, his family and friends, but also the economy of his country. It has many causes with a documented sequence of biochemical events leading to the destruction of the joint surface. There is as yet no successful cure, and even long term pain and dysfunction relief is not guaranteed by present day therapies. It is a condition which invites investigation by TENS therapy, because the possibilities of a safe, home based, electrical form of pain mediation that may help to reduce medication and increase joint function are persuasive.

CHAPTER 3

MECHANISMS OF PAIN MODULATION RELATED TO TRANSCUTANEOUS ELECTRICAL NERVE STIMULATION

Transcutaneous Electrical Nerve Stimulation is known to temporarily elevate the human pain threshold, thus improving the sufferer's ability to deal with his pain (Wolf & Gersh 1985). How TENS affects the pain threshold remains a subject for further experimentation.

3.1 The Function of Pain

Pain acts as a warning and as a teacher (Best & Taylor 1973). Tissue damage or a threat of impending damage activates local pain receptors, to notify the body to take action to avoid further injury (Watson 1981a). Without adequate pain reception, injuries can occur which lead to a degradation in lifestyle. Conditions which are characterised by reduced pain appreciation, such as hemiplegia, paraplegia and peripheral neuropathies, can be complicated by skin ulceration, limb fractures, joint dislocations, burns, pressure sores, infections and arthritis (Hopps 1964).

3.2 Action Potentials

Neurological studies on animals have shown that impulses travelling along a nerve fibre do so by electrical activity (Zimmerman 1976). Immediately a receptor is stimulated, an action potential is created by a wave-like flow of sodium and potassium ions in and out of the nerve cell. When a synapse is reached, the current is propagated by chemical transmitters across the synaptic space (Best & Taylor 1973), ensuring that the electrical impulse continues into the next structure. The frequency of generated action potentials is dependant on the intensity of the electrical stimulation. As the maximum frequency of a fibre is reached, additional afferent fibres are recruited to carry the impulse (Watson 1981a). Knowledge of the behaviour of fibres being stimulated by TENS should be relevant to predicting both the result, and the intensity needed to maintain a minimum effect (Wolf 1984).

3.3 Pain Receptors

The most common pain receptor is the free nerve ending, found in skin, ligaments, joints, muscles and periosteum (Kenshalo 1968). Specific receptors can have specialised functions such as taste, temperature, vision and hearing reception, but all receptors can appreciate pain if the intensity of the impulse is sufficiently great (Wolf 1984). The mechanism by which pain receptors are activated is thought to be the release of chemical substances which cause pain directly, or reduce local pain thresholds (Charl 1979). Naturally occurring substances such as 5 - Hydroxytryptamine, Substance P, bradykinin and some of the prostaglandins (natural occurring lipids) are thought to aggravate pain receptors, when released as a result of local injury. The subsequent current flow initiates a nerve impulse [Charl & Kirk (1975), Watson (1981b)].

Neurographic research has enabled peripheral pain impulses to be traced consistantly along the A-delta and C fibres (Torebjörk & Hallin 1974). Zimmerman (1976), Torebjörk & Hallin (1974) and Willis & Grossman (1973) currently accept a classification of human afferent nerve fibres. This is reproduced from Willis et al (1973).

Table 3.1 Presumed actions of afferent nerve fibres

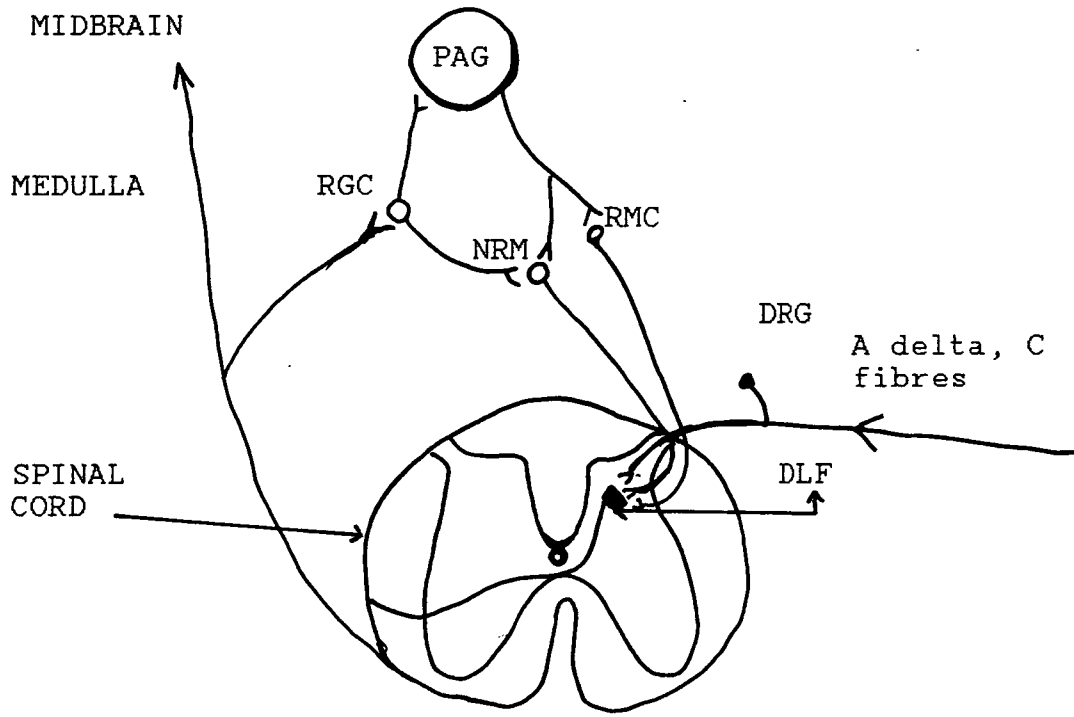
	Type	Group	Subgroup	Diameter (u)	Conduction Velocity (m/sec)	Presumed Function
	A	I	1a	12-20	72 - 120	Signal muscle velocity and length change.
	A	I	1b			Signal muscle shortening of rapid speed.
	A	II	Muscle	6-12	36 - 72	Signal muscle length changes.
	A	II	Skin			Convey information from touch receptors.
PAIN RECEPTOR	A delta	III	Muscle	1-6	6 - 36	Convey information from pain - pressure receptors.
	A delta	III	Skin			Convey information from pain, temperature or touch receptors.
	C	IV	Muscle	1	0.5-2	Convey information from pain receptors
	C	IV	Skin			Convey information from pain, temperature or touch receptors.

3.4 Ascending Spinal Pathways

Peripheral nerve fibres enter the spinal cord via the dorsal roots (Shealy, Mortimer & Reswich 1967). Actions of the ascending and descending tracts to the brain, have been established after electromyographical and surgical intervention in both humans and animals (Bishop 1980). The major spinal ascending pain pathway is thought to be the lateral spinothalamic tract (LSTT) (Watson 1981b). It carries thermal, pain, tactile, sexual and proprioceptive information (Noordenbos & Wall 1976), having both specific pain and sensory transmittor cells (Watson 1981b). Other ascending pathways, (such as the Dorsal funiculus) are known to carry pain information, but the LSTT is currently considered to be the main noxious pathway.

3.5 Connections at the Brain

3.5.1 LSTT fibres project initially to the ventroposterolateral nucleus of the thalamus, from where fibres carrying pain information project to the primary somatosensory cortex, an area concerned with the discriminative aspects of sensation such as localisation and identification of specific sensations and intensities. The medullary Reticular Formation, the Periaqueductal Grey, the Nucleus Rophe Magnus, and Nucleus Cuneiformis receive LSTT fibres at the midbrain level (Watson 1981b). These areas have connections to the medial thalamus, secondary somatosensory cortex and the hypothalamus, thus involving motivational, affective and autonomic responses to pain. The Periaqueductal Grey and Nucleus Rophe Magnus may be involved in a negative feedback loop (Basbaum 1980) (FIG. 3A) to suppress pain information segmentally.



DLF - Dorsolateral Funiculus
DRG - Dorsal Root Ganglion
NRM - Nucleus Raphe Magnus
RMC - Nucleus Reticularis Magnocellularis
RGC - Nucleus Reticularis Gigantocellularis
PAG - Periaqueductal Grey

Representative diagram only

FIG. 3A Negative Feedback Loop

3.5.2 The LSTT has connections to the Ascending Reticular Activating System which changes levels of consciousness and awareness. Cultural conditioning, posture, education, sympathetic nervous reactions and wellbeing may mediate pain reception via these pathways [Sternbach (1974), Watson (1981a)]. Cognitive influence in pain appreciation has yet to be fully explored. It is known that anxiety, stress, past experience, mental health, fatigue, and cultural attitudes alter the body's ability to perceive pain, but the method by which these inputs alter pain appreciation is still largely hypothesis. Descending cortical pathways may be involved in the feedback system where afferent input stimulates cortical output, which suppresses or stimulates afferent reception segmentally (Sternbach (1975). Expectations associated with TENS for chronic pain relief, especially in an osteoarthritic condition where other forms of pain relief may have failed, cannot be discounted as a reason for success.

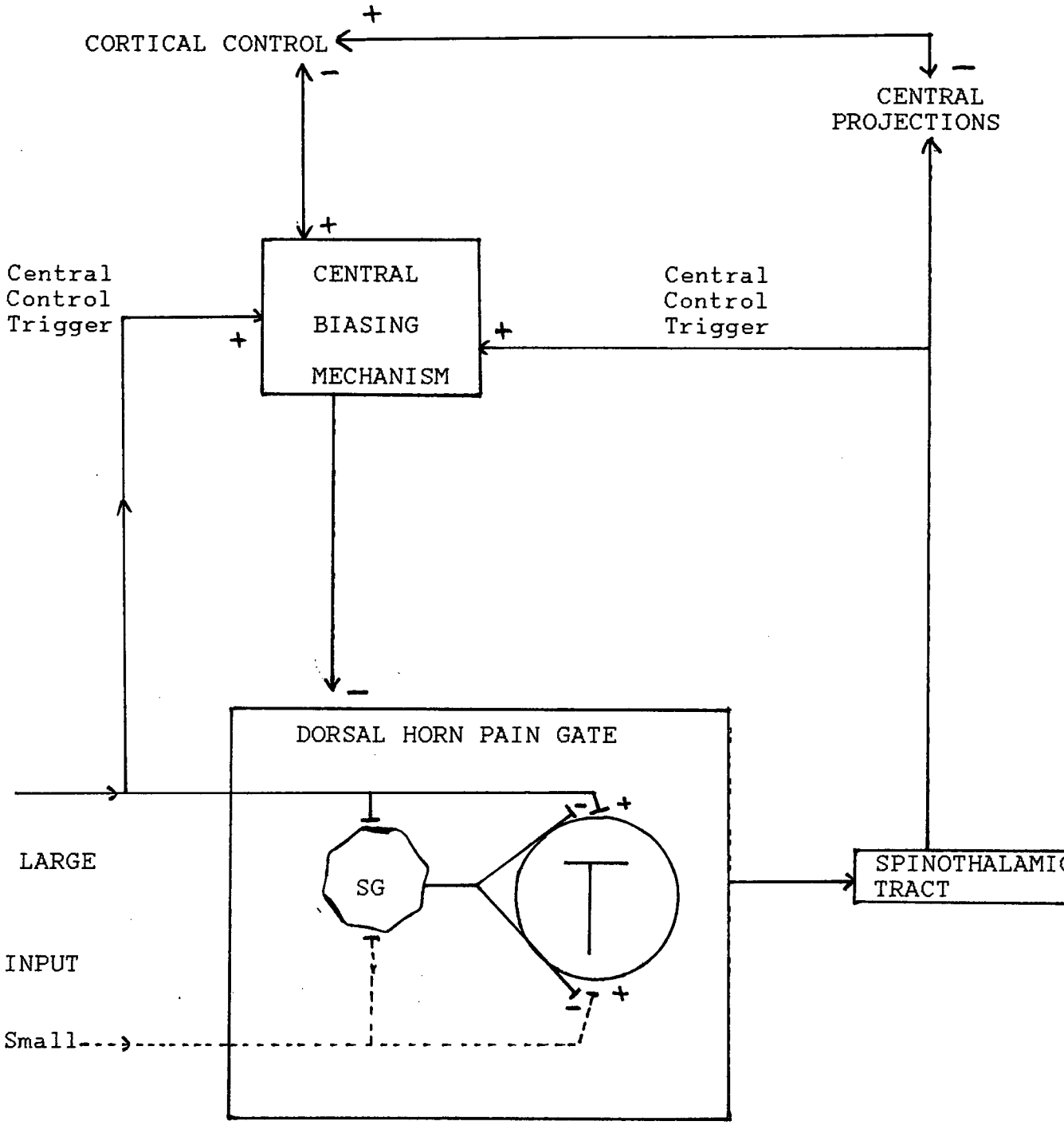
3.6 Theories of Neural Transmission of Pain

The efficacy of Transcutaneous Electrical Nerve Stimulation in pain relief may be explained by the following theories. However, Lundberg (1984) suggested, after reviewing the current TENS literature, that the effects of TENS may not be explained adequately by current knowledge of neurophysiology, anatomy or psychology. His views concur with those of Wolf & Gersh (1985): knowledge of TENS action may never be fully understood because of the complexities and individual eccentricities of the human body.

3.6.1 The "Pattern Theory" (Nafe 1929) hypothesises that sensation is not relayed by specific receptors, but rather by the pattern, frequency and intensity of impulses arriving in the brain. The pattern theory provides a possible basis for pain relief continuing after ceasing TENS; i.e. TENS may disrupt the "normal" chronic pain pattern to allow the establishment of a more acceptable pattern, by some other therapeutic modality such as exercise, massage or mobilisation. A return to "normal" may occur up to one hour after pattern disruption, unless a stronger, more dominant and permanent pattern is established in that time (Melzack & Loeser 1978).

3.6.2 The Pain Gate Theory (Melzack & Wall 1965) initially described "the constant and dynamic interaction between large diameter (A alpha, beta & gamma fibre) inputs and smaller diameter noxious (A delta and C fibre) inputs at the segmental level of the spinal cord". The Pain Gate theory has been confirmed by the identification of transmitter cells and interneurons in the Dorsal Horn, whose axons transmit pain sensations to the brain (Cervero & Iggo 1980). This theory possibly explains the immediate pain blocking effect of High Rate TENS.

3.6.3 The Central Biasing Theory, which evolved as a result of the Pain Gate mechanism, includes the possibility of influence in pain mediation by higher brain centres as well as spinal segmental inhibition [Melzack & Taenzer (1977), Melzack & Loeser (1978), Wall (1978)] (FIG. 3B). This mechanism may explain both the immediate and continuing action of High Rate TENS on chronic pain [(Ersek 1977), Melzack & Wall (1983)].



\+ excitatory

\- inhibitory

SG - Substancia Gelatinosa

T - Transmission Cell

Representative diagram only

FIG. 3B Central Biasing Mechanisms (Melzack & Loeser (1978) incorporating Melzack & Wall Pain Gate Theory (1965)

3.7 Endogenous Biochemical Theories of Pain Modulation

3.7.1 Research over the last fifteen years has explored the biochemical and neuropharmacological aspects of pain mediation. Acupuncture analgaesia, induced both by needle and Low Rate TENS, has been found to increase individual levels of endogenous opiates, [Levine, Gormley & Fields (1976), Hughes, Smith, Kosterlitz, Fothergill, Morgan & Morris (1975)], but the mechanisms and components of such opiate release remain areas of debate.

3.7.2 The discovery of endogenous opiates and their receptors in the Periacqueductal Grey and the Nucleus Rophe Magnus raised questions about the chemical control of the Spinal Cord Dorsal Horn afferent pathways by higher centres (Bishop 1980). Basbaum & Fields (1978) postulated that a negative feedback loop may involve neuromodulation by pharmacological substances as well as neural mechanisms (FIG. 3A). Electrical stimulation of the human Periaqueductal Grey and Nucleus Rophe Magnus has been shown to inhibit the transmitting actions of Dorsal Horn neurons (Giesler, Gerhart, Yezierski, Wilcox and Willis 1981). The clinical effects of such inhibition include increases in pain threshold over prolonged periods (Nolan 1987).

3.7.3 Opiate receptors have been found at all levels of the dorsal horn of the spinal cord. Binding endogenous opiates, (released via a descending modulatory system) (Basbaum 1980) to local opiate receptor sites at any spinal segmental level could explain a regional blocking mechanism that may complement segmental neural inhibition of pain impulses (Bishop 1980).

3.7.4 Peripheral synapses are also implicated in opiate blocking activity. Cervero & Iggo (1978) consider that intrinsic opiate receptors exist at most peripheral synapses, and can be activated or suppressed by chemicals that transmit pain. Substance P and serotonin are thought to be the main peripheral synaptic neuro-transmitters. When liberated as a result of local trauma, Substance P excites the neurons that are sensitive to pain, thus propagating pain impulses. In low doses, synaptic Substance P appears to inhibit pain appreciation by aiding release of local endorphins (Henry 1980).

Serotonin is a naturally occuring amino acid involved in temperature, pain, sleep, mood and appetite regulation (Mayer, Price & Rafii 1977). When released by local trauma, serotonin aggravates peripheral synaptic chemoreceptors. At brain and spinal cord synapses, however, serotonin supresses cells which receive pain input, thus enhancing release of endogenous opiates (Handwerker 1980). Adequate levels of dietary tryptophan are required to maintain serotonin manufacture in humans. Vitamin B3 (gained from eggs, meat, poultry and dairy products) must be eaten in sufficient quantities to maintain tryptophan levels. It is suggested that chronic pain may cause the depletion of serotonin stores by constant stress and/or inadequate diet (Shanks, Clement-Jones & Linsell 1981). Reduction in serotonin production may result in inadequate levels of endorphin production, thus altering pain thresholds (Mannheimer et al 1984).

3.8 Endogenous Opiates

Concentrations of intrinsic opiates in normal blood fluctuate in a circadian manner (Terenius 1979). Lowest baseline levels of plasma intrinsic opiates can be expected in the afternoon. Levels also vary according to the season; being lowest in the winter (Hughes et al 1984). Chronic pain subjects have been found to have lower CSF and plasma concentrations of endogenous opiates [Sjölund, Terenius & Eriksson (1977), Terenius & Wahlstrom (1975)]. Terenius et al (1975) suggests that this may be as a result of hypoactivity of the systems releasing endorphins, while Sjölund et al (1977) postulate that chronic pain patients may have a higher consumption of released endorphins.

3.8.1 Endorphins

Endorphins have been defined (Matsukura 1978) as long alpha- and beta- peptide chains existing in the CNS and peripheral synapses. Stimulation of the Periaqueductal Grey and Nucleus Rophe Magnus releases B-endorphin, which has behavioral consequences similar to those observed in morphine administration [Giesler & Liebeskind (1976), Liebeskind & Paul (1977)].

B-endorphin has been described as the longest acting and most potent of the endogenous opiates, (Tseng, Loh & Li 1976), being 30 times more potent on a molar basis than morphine. It is a 31 amino acid chain, an independant part of the pituitary hormone B-lipotropin. Kosterlitz (1979), and Ignelzi & Atkinson (1980) estimate its half life as at least 2 - 3 hours, as it is resistant to enzyme degradation. It is released by the pituitry gland in response to repeated stressful stimuli, and appears in the Cerebrospinal fluid within 30 minutes of such a stimulus commencing [Sjölund & Eriksson (1979), Akil et al (1978), Terenius (1979)]. Traces of B-endorphin have been found in the blood stream 24 hours after ceasing stimulation (Shanks, Clement-Jones & Linsell (1981). B-endorphin receptors at synapses (both peripheral and central) have been shown to be blocked by the administration of naloxone hypochloride, thus reversing endorphin mediated analgaesia [Sjölund & Eriksson (1979), Terenius (1979), O'Brien et al (1984), Hughes et al (1984)].

3.8.2 Enkephalins.

The enkephalin group of methionine- and leucine-enkephalin is composed of short chain (5 amino acid) peptides, found in the Periaqueductal Grey, Limbic System, Basal Ganglia, inter-neurones, Hypothalamus and Spinal Cord. Enkephalins have rapid enzymatic degradation, having half lives of less than one minute (Ignelzi & Atkinson 1980). Hughes et al (1975) suggested that enkephalins were weak morphine-like agonists, with leucine-enkephalin being half as strong as methionine-enkephalin. It does not appear that their receptors are blocked by naloxone (Clement-Jones, McLoughlin, Tombin, Besser, Rees & Wen 1980). Given descriptions of experimental data to hand, naloxone, administered within 15 minutes of TENS activity [Sjölund & Eriksson (1979), O'Brian et al (1984), Hughes et al (1984)] may not inhibit enkephalin activity simply because the enkephalins already have been broken down.

3.8.3 Other intrinsic opiates

Peptides with intrinsic opiate activity are still being discovered (Mannheimer et al 1984). Already, several with actions stronger than B-endorphin have been isolated; neurotensin, bombesin, dynorphin and angiotensin [Miller & Deyo (1980), Basbaum (1980)]. Increased levels of the hormones ACTH and B-lipotropin also have been found in CSF following stimulation of endorphin activity by repeated stressful stimuli.

3.8.4 Endogenous Opiates and TENS

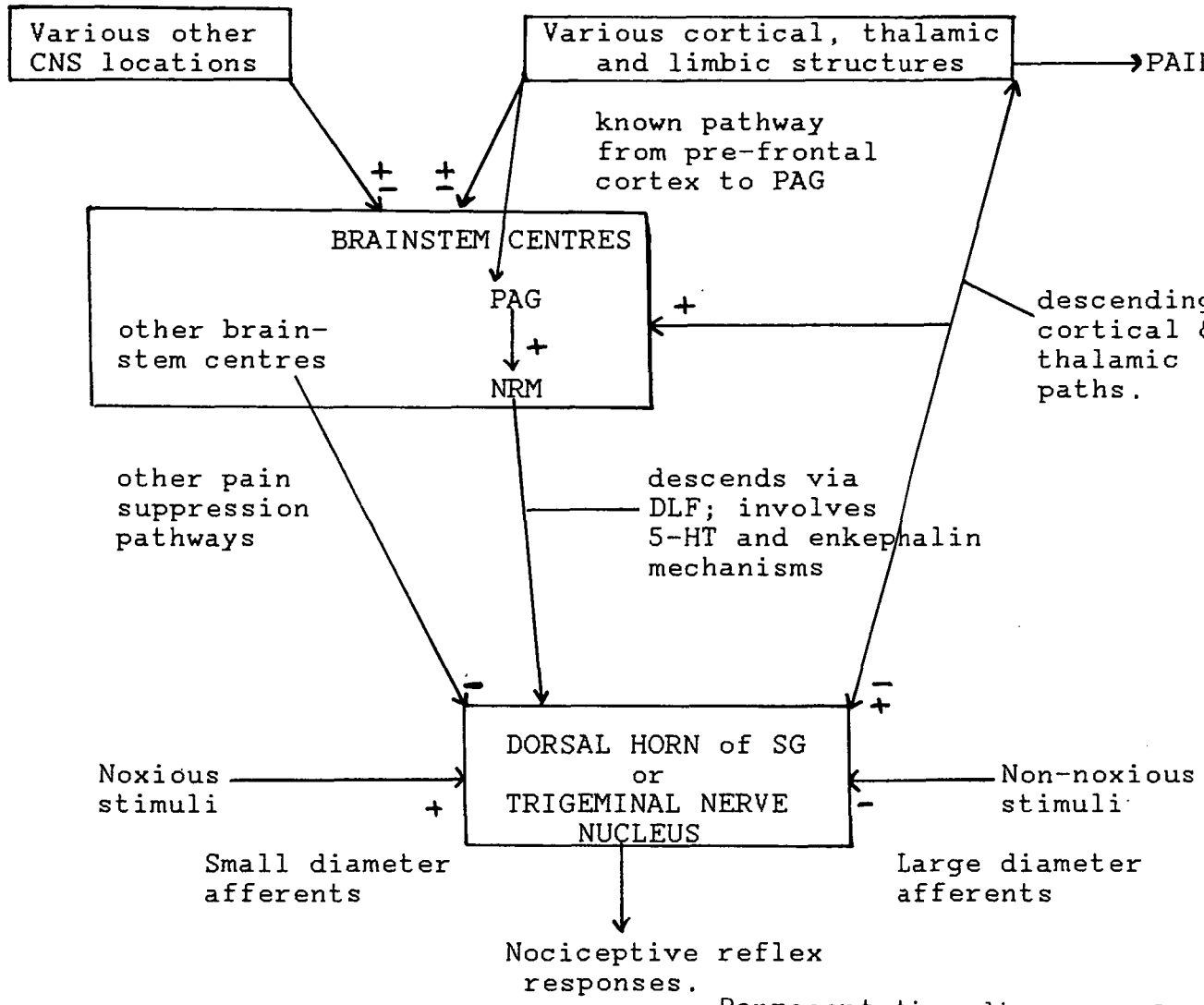
The discovery of increased B - endorphin levels in the CSF, brainstem and blood stream after Low Rate TENS application has been well documented [(Andersson, Eriksson & Holmgren (1973), Andersson & Holmgren (1975), Mannheimer et al (1984)], although dispute exists in the literature as to whether intrinsic opiates are stimulated by High Rate TENS application [Andersson & Holmgren (1978), Hughes et al (1984), O'Brian et al (1984)]. Sjölund & Eriksson (1979) and Clement-Jones (1983) found, after studies on experimental pain, that naloxone reverses the effects of Low rate TENS, but not High Rate TENS. Hughes et al (1984) suggest that the High Rate TENS effect (thought to be mainly a spinal nerve blocking action), may also be mediated by enkephalin production, but its half life may render it inactive before naloxone can block it. Understanding of this issue is far from clear.

3.9 The Effects of Naloxone Hypochloride

Naloxone hypochloride is a specific narcotic antagonist which has no morphine-like actions. It acts within two minutes of intravenous injection. Unfortunately, experimental differences with naloxone reactions on humans have been found to exist (Buchsbaum, Davis & Bunney 1977); humans with low pain thresholds obtain increases in pain perception from naloxone administration, while pain sensitivity decreases in patients with high pain thresholds. Naloxone may not block all intrinsic opiate receptors. Chronic pain sufferers differ from experimental pain subjects in that they have lower "normal" pain thresholds. The concentration amd composition of endogenous opiates stimulated by chronic pain may also differ from those found in single experimental pain experiments (Sjölund, Terenius & Eriksson 1977).

3.10 Conclusion

An impasse is evident in the argument of chemical versus neural pain pathways in TENS mediated pain relief (Bishop 1980) (FIG. 3C)



Representative diagram only

Legend:

CNS - Central Nervous System
DLF - dorsolateral fasciculus
NRM - nucleus raphe magnus
PAG - periaqueductal grey
\+ facilitatory influence
\- inhibitory influence
SC - Spinal Cord
5-HT - 5 Hydroxytryptamine

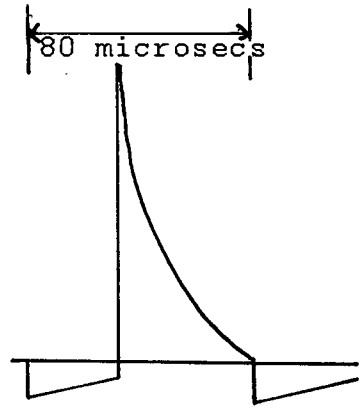
FIG 3C Chemical v. Neural Pathways in Pain Mediation

Lampe & Mannheimer (1984) describe the current pain transmission mechanism dilemma; "A proper mixture of chemical, electrical and neural events must take place at the synapse for inhibition or facilitation of nociceptive impulses to occur. The physiologic action of TENS can no longer be explained solely by the gate control theory, or by neuropeptide liberation. Clarification of the mechanisms by which the applications of High and Low rate Transcutaneous Electrical Nerve Stimulation to painful conditions may relieve pain is imperative to enhance the understanding of these powerful modalities". The concept of post TENS stimulation modulation of pain, possibly via descending cortico-spinal systems coupled with a physiological and /or narcotic spinal blocking mechanism, presents TENS experimenters with continuing future areas of research.

TRANSCUTANEOUS ELECTRICAL NERVE STIMULATOR DESIGN; VARIABLES OF APPLICATION.

4.1 Transcutaneous Electrical Nerve Stimulator Design

4.1.1 Transcutaneous Electrical Nerve Stimulator current is low voltage, direct current. Batteries are used to power the circuit, ensuring portability, and freedom from mains shock (Wall 1985). TENS current is composed of a biphasic balanced waveform with a nett zero current component (Medtronic Selectra Manual 1984). The Medtronic Selectra TENS alternately generates current from both electrodes. Three waveforms are currently on the market; spike wave, asymmetrical square wave and symmetrical square wave. Spike wave is the most economical of these; it requires battery power only to raise it to its peak, with a natural exponential fall-off. As TENS use for relieving symptoms of an osteoarthritic knee would be regular and longterm, the most economical waveform is desirable. Spike wave of pulse width 80 microseconds, (fixed) - set at half maximum amplitude with 500 ohms resistive load, is used in this experiment (FIG. 4A). This is the fixed spike wave shape for the Neuromod Selectra.



Representative diagram only.

FIG. 4A Spike Wave used for this experiment

4.1.2 TENS current can be delivered by a variety of electrodes. The most common electrodes presently used are made of carbon - rubber, with a silicone component to facilitate current flow. Electrodes can also be cut from inert metal, to any desired shape. A variety of jacks are available to apply electrodes in one or two channels. A contact medium is necessary to safely facilitate current passage; water soluble gel or wet sponge pads are most common depending on electrode choice. TENS electrodes can be self-adhaesive (using a chloride - free gum polymer interface), but the experimentation needed to establish safe user guidelines for these, is not well documented (Lampe & Mannheimer 1984).

4.2 The Choice of Transcutaneous Electrical Nerve Stimulator

The TENS chosen for this experiment was the Neuromod Selectra, manufactured by Medtronic Inc. (FIG. 4B). This choice was influenced by
1. Campbell's (1982) comparative research on TENS reliability,
2. the ability of this unit to produce both High Rate and Burst Mode TENS current at the push of a button,
3. the fact that machine's parameters closely adhere to the standards set by AAMI (1981),
4. the fact that the fixed narrow spike pulse width allowed the delivery of accurate High Rate TENS and Burst Mode train pulses. At this pulse width, pure Low Rate TENS would have been very uncomfortable, and adequate stimulation levels may not have been possible,
5. and by the fact that an earlier Medtronic TENS model was successfully trialed in experiments by Melzack, Vetere & Finch (1983), Hughes et al (1984) and McKelvy (1978).

Independent tests of pulse rate, wave shape and intensity increase by Hobart Medical Engineer, Mr T. Reagan of Medical Engineering Pty. Ltd. indicated consistent correlation between this unit's written parameters and its actual output. An advantage in using this unit was the digital display of intensity, which this author felt aided the placebo authenticity.

Scale 1 : 1

FIG. 4B Neuromod SELECTRA Transcutaneous Electrical Nerve Stimulator used in this Test.

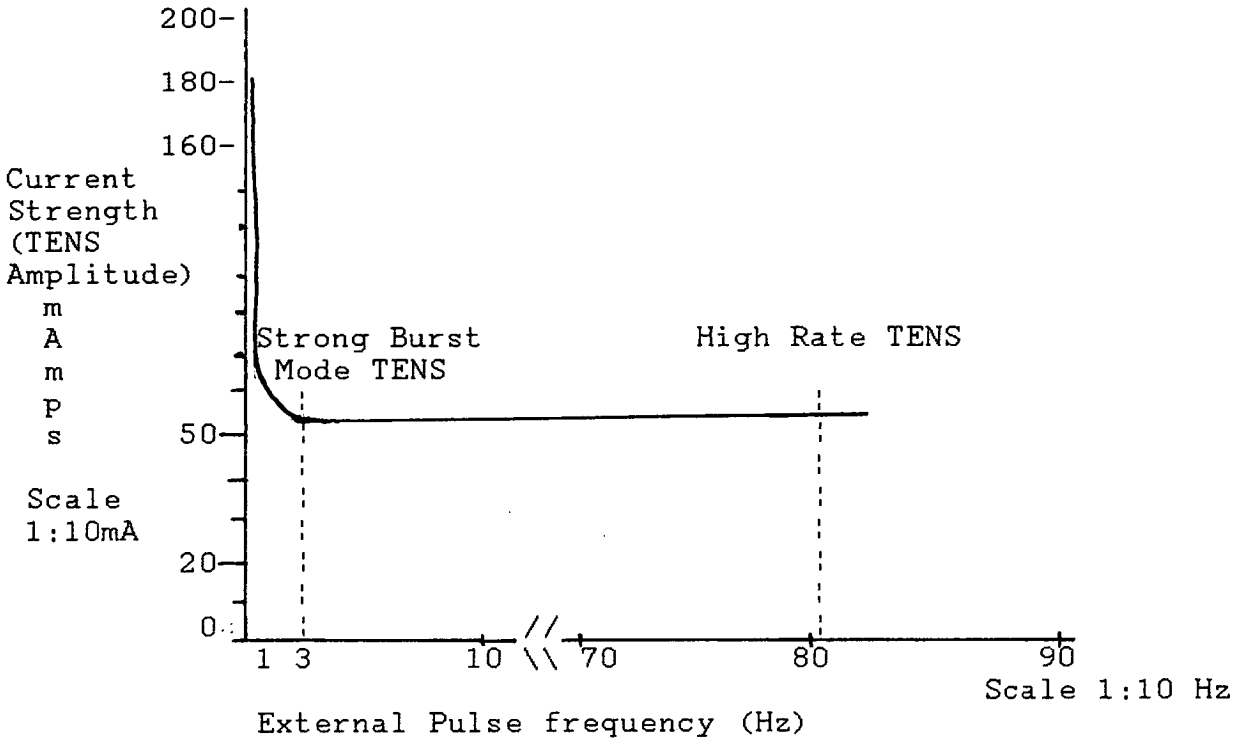


FIG. 4C Strength - Duration Curve showing stimulation intensities necessary for analgaesic levels of stimulation using High Rate and strong Burst Mode TENS.

4.3 The Accomodation Effect

As a nerve accommodates to a constant current, the potential difference experienced across the nerve due to the current flow no longer excites the nerve fibre. The potential difference will be maintained if the nerve is subjected to a sudden increase or decrease in applied external current. Scott (1969) says "a current which rises or falls suddenly in intensity is more effective in initiating an impulse than one which changes slowly. If the variation of current is gradual, there is time for accommodation to take place, and a greater intensity is needed to be effective.... A current that changes very slowly does not initiate a nerve impulse." In clinical TENS application, maintainance of perceived stimulation should occur by periodically increasing the intensity of the current (FIG. 4C).

4.4 Transcutaneous Electrical Nerve Stimulation in a Clinical Setting

4.4.1 TENS can be applied as a clinical assessment or treatment, or at home for regular pain relief. Assessing TENS actions in a purely clinical setting poses problems. Wolf, Gersh & Kutner (1978) suggest that clinical physiotherapists do not have the time or training to monitor TENS parameters in their everyday practice. "They need a definitive guide to settings that are successful for individual conditions". Wolf & Gersh (1985) and Mannheimer (1987) also suggest, that to stablish a fuller understanding of the effect of TENS current, objective measures of joint performance, changes in blood flow and in functional ability should be documented.

Although the experimentation is sparce, and this issue disputed, it must be noted that some writers consider that chronic pain patients often do not have an adequate diet, may not have normal amounts of exercise to metabolise what food they ingest and may suffer from endogenous depression [Falk 1982, Phillips (1983)]. Falk (1982) and Ward, Bloom & Friedel (1979) suggest that for the most successful application of TENS, a chronic pain sufferer's diet should be reviewed, (Vitamin B3 intake in particular), his energy expenditure monitored, and his mental state assessed to indicate his pain threshold and his level of depression. Mannheimer (1987) concurs, saying that the areas of diet, pain induced depression, and self motivation, are not well understood.

4.4.2 High Rate TENS Action on Osteoarthritis.

High Rate TENS (70 - 100 Hz) has been well documented as being effective for osteoarthritic conditions. Shealy, Beckner & Prieto (1974) and Ersek (1977) spoke of the Pain Gate Blocking effects of this TENS mode for chronic musculoskeletal pain, while studies on the effects of High rate TENS on osteoarthritis have been noted by Thorsteinsson et al (1977), Frampton (1982), Hughes et al (1984), Paxton (1980), Wolf et al (1981), Moore & Blacher (1983), Melzack et al (1983), Wolf & Gersh (1985). "TENS may be an important adjunct in the rehabilitation of arthritic patients, especially when joint replacement is not possible" (Wolf & Gersh 1985).

The efficacy of High Rate TENS on osteoarthritis of the knee is discussed by Mannheimer et al (1984), Smith et al (1983) Taylor et al (1981), Thurin, Meehan & Gilbert (1980) and Dougherty (1979). Although the effects of High Rate TENS as a pain relieving mechanism were indicated by these experiments, variations in application parameters, and pain relief results, suggested that further testing could be supported. In particular, the variation in length of pain relief experienced after stimulation ceases, continues to be unexplained. It is suggested that the most effective stimulation intensity is at tolerable levels, creating a constant tingling feeling radiating through the entire painful area (Mannheimer 1987).

High Rate TENS may mediate osteoarthritic pain by superimposition of stimuli, disruption of pain patterns and/ or endogenous opiate induction. Cyriax (1977) and Apley (1977) suggest that osteoarthritic pain can be separated into A - delta and C fibre componants, emanating from damaged bone, capsule, ligament, cartilage and periosteum. Nolan (1987) states, "it is still impossible to state categorically the exact actions of afferent fibres". Thus the pain mediating effects of High Rate TENS with respect to the specific pain of osteoarthritis are still unclear.

4.4.3 Low Rate or Burst Mode TENS Action on Osteoarthritis

Although acupuncture is well known for its chronic pain relief (Mann 1987), there appears to be little written about the action of either Burst Mode or Low Rate TENS on osteoarthritic conditions. Indeed, the action of these currents for any condition is poorly documented (Wolf & Gersh 1985). Although Low Rate TENS currents are recognised for creating intrinsic opiate stimulation, the length of treatment time and the intensity necessary for maximum results appear to have defused most interest. It has been established that Low Rate TENS increases B-endorphin levels in plasma and Cerebrospinal Fluid after 25 - 30 minutes [Sjölund & Eriksson (1979), Terenius (1979), Wilkes et al (1980)]. A slow rise occurs in pain threshold, which remains high for some hours, then slowly returns to normal (Mannheimer & Carlsson 1979), painting a picture of systemic time-release of a potent endogenous opiate.

Low rate TENS currents (2-3 Hz) have a different action from High Rate TENS. This current is not selectively seeking large fast acting afferent fibres. It requires the involvement of neuromotor junctions, in order to stimulate endorphin production by repeated stressful stimuli. Possible direct neural effects of Low Rate TENS have been poorly addressed. Lundberg (1984) allows that while Low Rate TENS is known for its potent chemical pain mediation, it may also create neural pain mediation via direct action on pain receptors. There has been little experimentation done in this area, and pain relief mediated by Low Rate TENS has been mostly explained by the half life of intrinsic opiates rather than by any other mechanism.

Mannheimer (1987) notes that the intensity, the strong muscle contraction and the length of Low Rate TENS necessary for pain mediation, may render it unsuitable for application to an inflammed, painful joint. He promotes the use of the recent Low Rate TENS adaptation - strong Burst Mode TENS, for patients with chronic joint pain. While low intensity Burst Mode TENS can be used as to produce a High Rate TENS effect, the strong, pulsing application of this current mimics the effects of Low Rate TENS. It causes the strong rhythmic muscle contractions necessary to produce the Low Rate TENS-type stimulation of the endogenous opiate mechanisms, but does so at more comfortable intensities. It can be tolerated well for the 30 or more minutes necessary to trigger intrinsic opiate mechanisms (Sjölund & Eriksson (1978).

Burst Mode TENS used in this study comprises train pulses generated at 3Hz, with an internal pulse frequency of 80Hz (Medtronic 1984). The peripheral nerves recognise the carrier wave as Low Rate pulses, while the skin allows passage of the higher frequency train pulses, with less resistance than to single Low Rate pulses (Wolf 1984). The muscle spindles also react at lower thresholds to the train of faster pulses than it does to the slower single Low Rate pulse. Thus the muscle contraction necessary for intrinsic opiate action is able to be stimulated by lower current amplitudes than for pure Low Rate TENS, in a more comfortable application, yet producing the same physiological and biochemical effects [Mannheimer (1987), Eriksson et al (1979)].

Table 4.1 Initial Stimulation Parameter Settings of Low Rate and strong Burst Mode TENS currents (reproduced Mannheimer & Lampe 1984)

	Low Rate TENS	Strong Burst Mode TENS
Frequency	1 - 4 Hz	2 - 4Hz
Pulse duration	150 - 250 millisec	100 - 200 millisec
Amplitude	At or above tolerance, producing strong, rhythmic muscle contractions. 30 - 80 mA	To tolerance, producing strong, rhythmic muscle contractions. 30 - 60mA
Action	On intrinsic opiate mechanisms after 30 mins, and possibly local peripheral synapses.	

The use of tricyclic antidepressants (such as amitriptyline) in the treatment of osteoarthritic pain is a current trend [Calabro (1986), Altman (1986)]. One of the roles of this class of drug is to stimulate the central release of endogenous opiates. As Low Rate TENS has this effect, it seems reasonable that Low Rate TENS (or adaptations of it) may also be successful in producing viable pain relief for osteoarthritis sufferers. "The role of clinically induced endorphins and enkephalins in relief of osteoarthritis pain is recognised, but as yet unclarified" (Altman 1986). Ward, Bloom & Friedel (1979) and Falk (1982) suggest that Low Rate TENS effectiveness could be enhanced by the prescription of trycyclic antidepressants. As High Rate TENS production of endogenous opiates is unsubstantiated, an area of research using TENS frequencies and trycylic antidepressants may be indicated.

Low Rate (and its adaptation Burst Mode) TENS cause changes in tissue as a result of its stimulation of muscle fibre and blood supply. The physical effects of its application to local tissues have not been well addressed in the literature. The pain mediating effect of muscle activity, change in local blood supply, and change in chemical composition around joints as a result of muscle activity are not well researched. Altman (1986) suggests that one of the possible mechanisms of pain production from an osteoarthritic knee is the synovial membrane inflammation. Although such inflammation may be directly related to mechanical irritation from joint laxity or loose fragments, it may also be due to chemical changes "such as the release of collagen, proteoglycans, or enzymes from the damaged cartilage, which in turn may activate synovial mediators of inflammation, such as prostoglandins or interleukins". It seems possible that local actions of induced endogenous opiates via Low Rate TENS mediation may block or reverse some of these toxic changes [Inversen (1979), Handwerker (1980)].

Table 4.2 A Physiological Comparison of High and Burst Mode TENS

High Rate TENS	Strong Burst Mode TENS
1. Primarily large (proprioceptive) afferent fibre stimulation (narrow pulse width and high pulse rate).	1. Primarily small (pain) afferent and (motor) efferent fibre stimulation (narrow pulse width and low pulse rate).
2. Produces a comfortable parasthaesia without muscle contraction. (not as effective below perception level).	2. Produces a strong muscle contraction. Effectiveness increased by highest tolerable contractions.
3. Fast onset (accommodation occurs needing amplitude readjustment).	3. Slow onset (little or no accommodation. May activate summation mechanisms).
4. Relatively short aftereffect	4. Relatively long aftereffect
5. Little or no endogenous opiate liberation.	5. Endogenous opiate liberation
6. No reversal by naloxone	6. Reversal by naloxone
7. Primarily local CNS effects	7. Primarily higher CNS effects
8. Stimulation by surface electrodes in segmentally related regions is most effective.	8. Stimulation by surface electrodes in segmentally related myotomes is more effective.

4.5 Parameters involved in TENS Application for Osteoarthritic Knees

Research into the specific variables in TENS therapy for osteoarthritic knees is far from completed. Wolf et al (1981) warned "that the lack of specific investigation into optimum electrode placements and stimulation parameters casts doubt upon the efficacy of TENS". Factors such as current flow, skin / electrode impedance, inter-electrode distance, frequency of stimulation, pulse shape and width, length of treatment time and differences in body part reactions to TENS remain largely unexplored. Grau (1981) suggests that frequency, intensity and duration of current are critical to analgaesia production. Mannheimer & Carlson (1979) suggest "TENS gives pain relief in a number of conditions. The underlying mechanism is not clear. Consequently the choice of optimal stimulation variables must be based on clinical experience." Wolf & Gersh (1985) urge further study. "Specific electrode placements and stimulation characteristics must be evaluated in relation to specific disease entities, to establish more effective treatment protocols".

4.5.1 Length of the Experiment

The length of this experiment was indicated by recent studies. [Mannheimer & Lampe (1984), Thurin et al (1980), Dougherty (1979), Smith et al (1983), Sjölund & Eriksson (1976)]. 30 minutes Burst Mode TENS is necessary to stimulate endomorphin production. Although blocking neural pathways occurs immediately with High Rate TENS, longer applications of High rate TENS may be necessary to stimulate more lasting forms of analgaesia (Lundberg 1984). As one of the aims of this experiment was to establish TENS as a viable form of post stimulation analgaesia for osteoarthritic pain, it was felt that 30 minutes stimulation was necessary to produce a definitive result.

4.5.2 Stimulation Levels used in This Experiment

4.5.2.1 High Rate TENS

Studies by Walmsley & Flexman (1979), and Andersson et al (1976) established that experimental High Rate TENS produces immediate pain relief over a range of intensities. Intensities set from "just above sensation threshold" to "at and above patient tolerance" all produce pain relief (Andersson et al 1976). As there was no necessity to increase the discomfort of the subjects in this trial, the commonly accepted stimulation level of High Rate TENS application has been used for this experiment. Mannheimer (1987) defines it as "a tolerable tingling (pins and needles) sensation (without muscle contraction) perceived throughout the area of the pain".

4.5.2.2 Burst Mode TENS

While early research [Andersson & Holmgren (1975), Holmgren (1975)] suggested that the intensity of Low rate TENS must be uncomfortable in order to stimulate increases in endogenous opiate production, later development of Burst Mode TENS to mimic a Low Rate current indicates that tolerably strong, repetitive stimulation producing muscle contraction will also create prolonged pain relief [Fields & Basbaum (1978), Fields & Andersson (1978)]. Again, there was no need to increase the discomfort of the subjects participating in this study, so the Burst Mode TENS option was used in preference to a pure Low Rate TENS application. Maintaining a strong muscle contraction around the knee was essential throughout this TENS application; such stimulation has been shown to produce a sustained neuronal discharge to the Nucleus Rophe Magnus and the Periaqueductal Grey (Eriksson et al 1979).

4.5.3 Electrode Placement

The type and extent of pain is the common guide to electrode placement for osteoarthritic knee treatment, although the position and size of electrodes, the type of electrode used and the direction of current flow are not often noted in the literature. While Low Rate TENS electrodes are usually placed over acupuncture points (see Section 5.2), High Rate TENS electrode placements for specific conditions [Wolf & Gersh (1985), Lampe (1984)] seem no more defined than those developed with early TENS therapy [Shealy (1974), Ersek (1977)]. The prevailing criteria seems to be that the electrodes be placed in such a way as to stimulate the local cutaneous nerve supply, and to cover as much as possible of the painful area. Mannheimer (1978) contends that there is a basic problem when deciding on common stimulation points; "using the same specific electrode placement sites for different patients with similar conditions is not always effective." He suggests that the selection of stimulation sites depends on "an awareness of etiology, location and character of the pain", and as this is individually mediated, it may be at this stage where research can no longer be specific.

Linzer & Long (1976) and Laitinen (1976) suggest that electrode placement is the most important parameter in TENS mediated pain relief. Several methods of application are demonstrated in the literature for applying High Rate TENS electrodes for the relief of osteoarthritic knee pain.

1. Painful skin sites were used by Berlant (1984), Melzack (1976), Melzack et al (1977), Smith et al (1983) and Taylor et al (1981).

2. Superficial points on peripheral nerves around the knee were used by Smith et al (1983), Dougherty (1979) and Mannheimer et al (1984). Those chosen were medial crural nerve, anterior femoral cutaneous nerve, lateral femoral cutaneous nerve and posterior femoral nerve.

3. Piggybacking multiple electrodes to stimulate wider cutaneous areas and deeper nerve plexuses is suggested by Mannheimer (1978), Wolf, Gersh & Kutner (1978) and Medtronic (1982).

4. Acupuncture points for relief of osteoarthritic knee pain are described by Mannheimer et al (1984), Smith et al (1983) and Thurin et al (1981). Points on Spleen, Gall Bladder, Kidney, Urinary Bladder and Liver meridians were used to stimulate the knee.

5. Motor points, sites which are characterised by high electrical conductance and low skin resistance (Goodgold & Eberstein 1978), are suggested by Medtronic (1982) and Mannheimer & Lampe (1984). Electrode placement over Spleen 10 coincides with a motor point of Vastus the medialis muscle, while Urinary Bladder 40 is a motor point of the Popliteus muscle.

4.5.4 Electrode Placement Choice for This Experiment

4.5.4.1 The choice of acupuncture points for electrode placement in this study was made because

1. acupuncture points make clinical anatomical placement better defined, and thus able to be reproduced,

2. by applying High Rate and Burst Mode TENS current to the same anatomical points it was hoped to maximise any analgaesia via the endomorphin mechanisms of the two TENS frequencies.

It is acknowledged that the electrodes used in this study would stimulate not only the exact acupuncture point, but also the other anatomical issues noted in 4.5.4.2, because of the electrode size. The electrodes available on the present TENS market do not allow stimulation of acupuncture points alone. Manufacture of smaller and safer surface electrodes is to be encouraged.

4.5.4.2 The acupuncture points chosen for this study are Spleen 9, Spleen 10, Urinary Bladder 33 and Gall Bladder 40. A description of the acupuncture points used in this experiment is found in Section 5.3. These points represent

1. points on four sides of the knee, thus providing irradiation of the entire joint (Ersek 1977),

2. common painful spots found in osteoarthritic knees [Taylor et al (1981), Berlant (1984)],

3. two motor points of muscles around the knee (Gunn 1976), and

4. stimulation of the cutaneous nerves which surround the knee (Gray's Anatomy 1972).

4.5.5 Current Flow

Parallel current flow between two channel electrode placements is discussed in Mannheimer (1978), Wolf, Gersh & Rao (1981), Mannheimer & Lampe (1984), Mannheimer (1987) and Medtronic literature (1982, 1984). Parallel currents are inherent in the original TENS theory and have been well documented (Shealy 1974). The current runs in the same direction as the cutaneous nerves that supply the knee. As is illustrated in FIG. 4D, current runs between Spleen 9 and 10 electrodes, and between Urinary Bladder 40 and Gall Bladder 33.

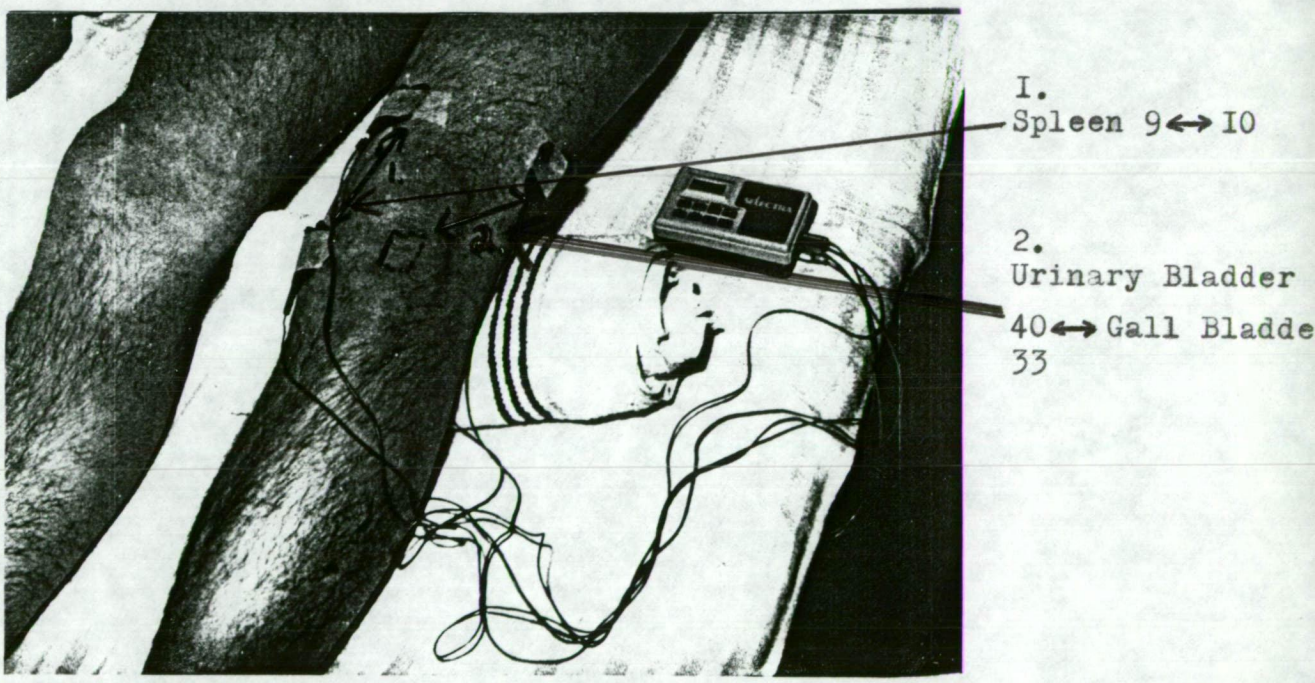


FIG. 4D Current Flow in this Study

4.6 Conclusion

The choices presented to those who prescribe and apply TENS for the relief of chronic osteoarthritic knee pain are abundant. What provides the most successful pain relief will be informed combinations within the available parameters. Advances in TENS technology may provide even more parameters from which to choose. It is vital that those who use this powerful modality understand the rationale of each of the parameters involved in TENS application, and are able to apply them to individual conditions.

ACUPUNCTURE POINTS: APPLICATION FOR OSTEOARTHRITIC KNEES

5.1 Acupuncture has enjoyed increasing Western world acceptance, following cultural exchanges resulting from the Chinese Cultural Revolution. Despite continuing scepticism from many traditional sections of the medical community (Mann 1973), acupuncture and/or use of acupuncture points is practiced successfully by doctors and paramedics for many painful and irritating conditions (Levine, Gormley & Fields 1976). Although acupuncture has been used succesfully in acute pain relief like headache (Melzack, Stillwell & Fox 1977), and childbirth (The Academy of Traditional Chinese Medicine (1975), it is suggested that acupuncture may have its greatest impact on modern medicine in the treatment of chronic pain [Mann, Bowsher, Mumford, Lipton & Miles (1973), Mann (1987), Melzack & Fox (1976)].

The characteristics of acupuncture points suggest that their use in all clinical applications of TENS is strongly indicated; the common acupuncture points are well documented, they are easily identified and reproduced, they overlie many superficial nerve roots and sensory nerve endings, and they have a high correlation with established medically used trigger and motor points.

5.2 Use of Acupuncture Points in TENS Applications for Osteoarthritis

Despite being described by Smith et al (1983) in a High Rate TENS experiment for osteoarthritic knee pain, acupuncture points are not the presently accepted choice for electrode placement for High Rate TENS administration to osteoarthritic joints. Because of the traditional understanding that High Rate TENS works mainly on the Pain Gate (Mannheimer & Lampe 1984), electrode placements are usual over spinal nerves, nerve pathways, painful spots, motor points or trigger points. Because it evolved from the theory of acupuncture, Burst Mode (or Low Rate) TENS must be applied to acupuncture points for the successful relief of chronic pain [Anderson, Eriksson & Holmgren (1973), Andersson & Holmgren (1975), Melzack (1976), Wolf & Gersh (1985)].

The acupuncture points used for Low rate TENS administration have been strongly correlated with trigger points and cutaneous nerve pathways used for High rate TENS administration [Mann (1987), Gunn & Milbrandt (1975), Melzack, Stillwell & Fox (1977)]. It seems logical to question whether High rate TENS, applied over traditional acupuncture points, may also stimulate the endorphin activity, currently associated with Low Rate TENS and acupuncture. Paxton (1980) suggests that wide-spread maximally effective use of TENS for chronic pain may require another 30 years study and education, while Altman (1986) suggests that "the role of clinically induced endorphins and enkephalins (via pharmacological or electrical agencies) in relief of osteoarthritic pain is yet to be clarified". It appears that the issue of endorphin production via TENS current over specific electrode placements is still to be fully addressed.

5.3 Acupuncture points Chosen for This Study

Descriptions of the points are reproduced from Essentials of Chinese Acupucture (1980) and Mann (1987).

5.3.1 Yinlingquan (Spleen 9) (He - Sea Point) (FIG 5A)

(Represents trigger point of medial aspect knee (Scott 1969)).

Location: On the lower border of the medial condyle of the tibia, in the depression between the posterior border of the tibia and gastrocnemius muscle.
Vasculature: Anteriorly, great saphenous vein, genu suprema artery. Deeper, the posterior tibial artery and vein
Innervaton: Superficially, the medial crural cutaneous n., Deeper, tibial n.
Indications: Abdominal distension, oedema, jaundice, diarrhea, incontinence, dysuria, pain in the knee.

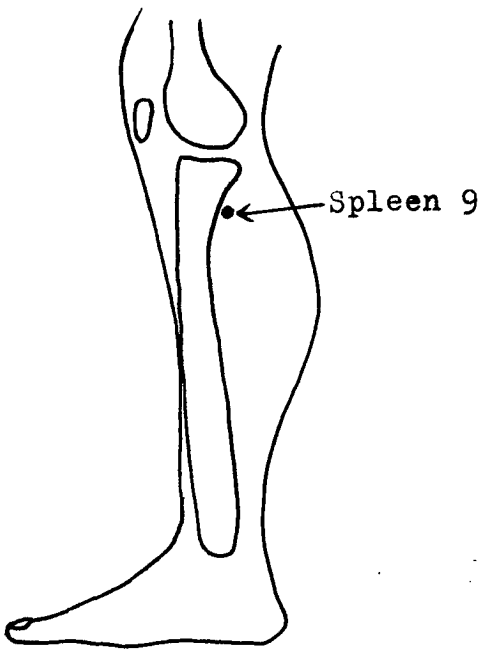


FIG 5A Spleen 9

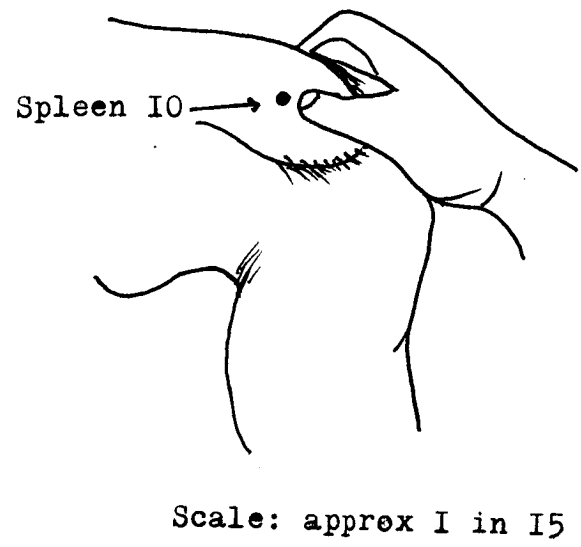


FIG 5B Spleen 10.

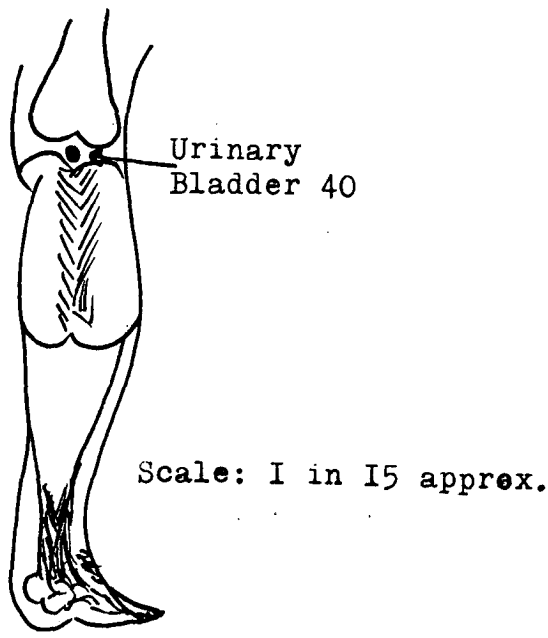
5.3.2 Xuehai (Spleen 10) (FIG 5B)

(Represents motor point of Quadriceps vastus medialis (Scott 1969)).

Location: When the knee is flexed, the point is 4 cm above mediosuperior border of the patella, on the bulge of the medial portion of quadraceps muscle. Cup R hand to the patient"s L knee, Thumb on the medial side - the point is at the tip of the extended thumb.
Vasculature: Muscular branches of femoral artery and vein
Innervation: Ant. Femoral cutaneous n., muscular branch fem. n.
Indications: pain in anterior / medial aspect thigh and knee

5.3.3 Weizhong (He - Sea Pt) (Urinary Bladder 40) (FIG 5C)
(Represents trigger point at posterior knee (Scott 1969)).

Location: Midpoint of transvese crease of the popliteal fossa, between tendons of biceps femoris muscle and semitendinosis m. Locate in prone or with a flexed knee.
Vasculature: Superficially - femoropopliteal vein, popliteal vein, popliteal artery.
Innervation: Posterior femoral n., tibial n.
Indications: Low back pain, motor impairment of hip, contraction of tendons of popliteal fossa, motor impairment and pain in lower extremities, muscular atrophy.



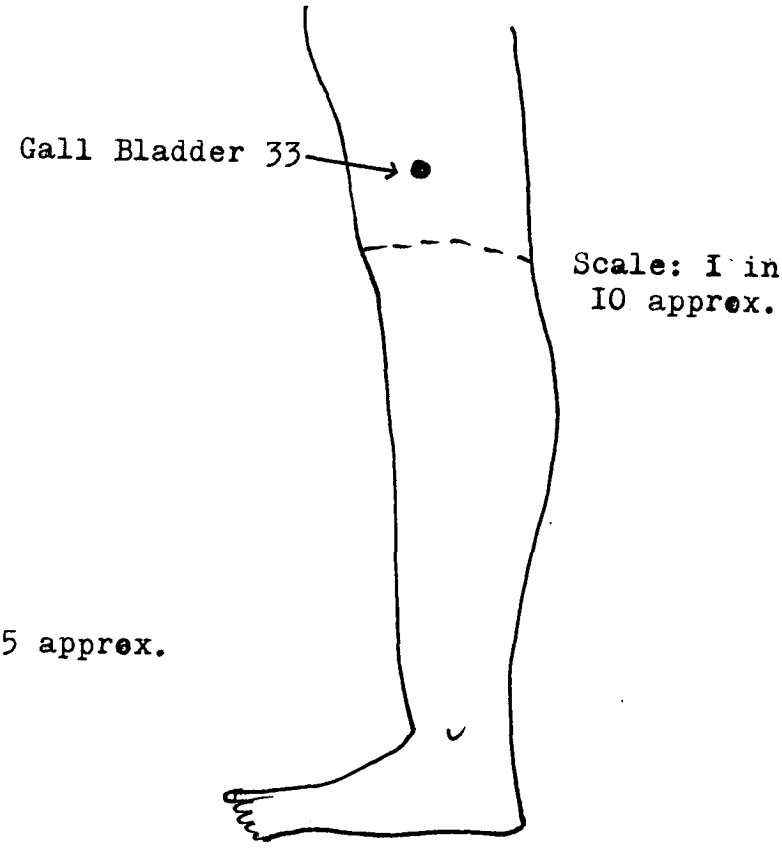


FIG 5C Urinary Bladder 40

FIG 5D Gall Bladder 33

5.3.4 Xiyangquan (Gall Bladder 33) (FIG 5D)
(Represents trigger point lateral aspect lower femur (Scott 1969)).

Location: Knee flexed, point is 6 cms above head of fibula lateral to the knee joint, in the depression between tendon of biceps femoris m. and the femur.
Vasculature: Superior lateral genicular artery.
Innervation: Terminal branch of lateral femoral cutaneous n.
Indication: Pain and swelling in the knee, contracted tendons in the popliteal fossa, numbness in the leg.

SUBJECTIVE AND OBJECTIVE ASSESSMENT OF OSTEOARTHRITIC KNEES

6.1 Chronic Osteoarthritic Joint Assessments

Sufferers from osteoarthritis of the knee experience discomforts that are difficult to measure experimentally. To gain a full appreciation of the effects of TENS on osteoarthritic symptoms, all areas of osteoarthritic dysfunction must be examined. Osteoarthritic pain has been mentioned already. Stiffness also is a well documented problem of osteoarthritis [Altman (1986), Calabro (1986)], as is muscle dysfunction, joint swelling and decrease in range of movement (Darby 1983). Pain, stiffness, joint swelling and range of movement may be inter-related; a change in one may create a change in another. For TENS to be proven effective for relief of chronic osteoarthritic symptoms, wider subjective and objective measures must be taken.

Sternbach (1976) suggests that "pain assessment based entirely on the patient's description of pain is a procedure that invites contamination and confounding by both psychological and sociocultural factors." Procacci, Zoppi and Maresca (1978) express wariness of sensory matching techniques because of the extra variables and psychological reactions caused by externally induced pain. Von Knorring, Almay & Johansson (1978) suggest that a patient with chronic pain may have a different reaction to TENS than one with no pain, because of changes in personality and expectation. Werner (1980) illustrates two components of pain; "the interpretive/ emotional component and the localizing/sensation component" that must be addressed to gain full insight into individual pain states. By dealing with subjects with chronic pain, as this study did, it was thought that the emotional component may be controlled; the chronic osteoarthritic knee would have limited the subject's lifestyle, created an expectation of pain and limitation in joint function at some stage in each day, and induced caution in attempting any physical exertion that may stress the knee (NH & MRC Report 1988).

TENS literature on chronic osteoarthritic pain to date has been limited by the measurements taken. Descriptive and/or numeric pain assessments, and varied indices of functional activity were the only measurements taken in the TENS and osteoarthritis literature reviewed for this study. TENS is considered to have been effective if it decreases the subject's immediate pain measurement by 50 percent. [Hansson & Ekblom (1983), Salar, Job, Minguino, Bosio & Trabucci (1981)]. Wolf & Gersh (1985) are critical of TENS literature to date: "most investigators still rely on the patient's report of pain to establish the efficacy of TENS treatment". Physical measures such as "joint range, strength, muscle girth" would enhance "established objective evaluation of TENS efficacy".

6.2 Measurements For This Study

When considering the measurements required by this study, several factors had to be considered:

1. the time taken to administer the test,
2. the degree of difficulty experienced by the subjects when completing the reqirements of the assessment,
3. the need to control the degree of complexity of this study,
4. the limitations of both the study and the measurements, and
5. the reliability of the measurements (see Section 6.2.2.1).

This study measured pain and stiffness (immediately and over time), and immediate post test knee circumference and range of movement to validate the effects of the TENS frequencies on osteoarthritic knees. Measurements taken were:

1. a quantitative measurement of a change in pain state,
2. a report of pain relief over 24 hours,
3. a quantitative measurement of change in stiffness state,
4. a report of stiffness relief over 24 hours,
5. objective circumference measurement immediately pre/post test,
6. and objective range of movement measurement immediately pre/post test.

6.2.1 Subjective Measurement - Pain and Stiffness

A number of choices were available in the area of subjective assessment of pain. This study included a measurement of stiffness as well as pain, although there is no precedent in TENS literature for measuring this. Stiffness is not necessarily a subjective assessment, although neither does it fall into an objective category. Measuring it subjectively highlighted clinical anomalies. Some joints were stiff only in one range, while others were stiff only at certain times of the day. To clarify this issue, subjects were asked for a measure of whether their immediate overall stiffness had improved with TENS current, and if so, by how much. It was felt that by conducting the measurements over 24 hours, variations in patterns of stiffness could be addressed.

The choices for assessing pain range from descriptive questionaires [Melzack (1975), Tursky (1976)], body diagrams (Kopala & Matassarin-Jacobs 1984), sensory matching (pain to pain), [Kast (1966), Sternbach (1974), sensory matching (pain to colour) (Stewart 1977), to Descriptive Pain Indexes [Ingham (1969), Stewart (1977)]. Several mutifaceted choices are available which are supposed to completely address the chronic pain state [Mannheimer & Lampe (1984), Hendler, Viernstein, Gucer & Long (1981), Shealy & Shealy (1976)]. These pain assessment methods were considered too detailed and too time consuming for the needs of this experiment.

The confines of the study, the need for quantitative measures of both immediate pain and stiffness change, and simplicity of application directed the choice to scale measurement. Although it may have been easier for the subjects to use Descriptive Scales [Stewart (1977), Melzack (1975), Johnson (1972)] or Numeric Scales (Stewart 1977), the choice for the Absolute Visual Analogue Scale was made because of its greater validity (see Section 6.2.2.1).

6.2.2 The Absolute Visual Analogue Scale

A pure line scale was used in this experiment. No graphic rating was indicated, and two scales were used side by side to describe pain and stiffness. The only descriptions used were "No Pain (Stiffness)" at the left (bottom) of the scale, and "Pain (Stiffness) as bad as you have ever had it" at the right (top). The subject marked his pain and stiffness estimations along vertical 10 cm lines, with readings taken with an accuracy of one decimal point (Scudds 1983) (FIG. 6A).

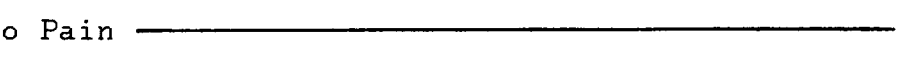


FIG. 6A Absolute Visual Analogue Scale

The vertical configuration used in this study (seen in Appendix 2) was chosen with regard to argument as to preferred alignment [Stewart (1977), Downie, Leatherman, Rhind, Wright, Branco, Anderson (1978), Scott & Huskisson (1976)]. It was hoped that ease of using the scale may be enhanced by its likeness to a thermometer (Zussman 1986).

It was accepted that it may be difficult for some patients to transfer their pain and stiffness sensations into visual and linear dimensions, and that the abstract quality of the visual analogue scale may frustrate some test subjects (Kremer, Atkinson & Ignelzi 1981). The subject's visual and motor coordination was essential, as was "the ability to place a mark where he (the patient) intends to put it." (Revill et al 1976). Potential subjects who suffered from autism or dementia were considered unable to use this scale (Merskey 1974).

6.2.2.1 Advantages of AVAS

The advantages of the scale as accepted by this author are:

1. It is easily administered (Zussman 1986) and therefore suitable to a clinic application. Its simplicity and lack of descriptive direction made it an ideal experimental assessment tool.
2. Pain and stiffness estimation can be reproduced and is reliable over time (Revill et al 1976).
3. Statistical inferences can be drawn because the scale is not tied to graphic descriptions, thus avoiding the inequal internal comparison (Stewart 1977).
4. The 10 cm line is acceptable for statistical error (Revill et al 1976).
5. The scale is sensitive to pain and stiffness change (Clarke & Spear 1964).
6. It "does not force quantum changes in pain intensity as occurs with category scales" (Ohnhaus & Adler 1975).
7. The patient choices are not limited by
   a) lack of appropriate descriptive words (Price, McGrath, Rafii & Buckingham 1983) as in McGill-Melzack Study.
   b) having to read and understand English
   c) having to engage in, or rate Activities of Daily Living for a total pain assessment. This limits fear, anxiety, disinterest, clinically produced pain and associated musculoskeletal disorders which may intensify reactive pain (Linton 1985).

6.2.2.2 Disadvantages of AVAS

The disadvantages found with the scale in this study were:

1. It did not address pain differentiation ability between subjects. Some asked whether the scale represented the soreness or the ache felt in the affected knee joint.
2. The scale did not allow for different pain intensities depending on subject's position; eg it was noted that pain varied depending on posture, weightbearing, clothes being worn and the weather. This was addressed by taking all AVAS measurement in weight-bearing, before and after the test, with the knee unclothed.

6.2.3 Verbal Reports of Pain and Stiffness

Verbal reports of length of pain and stiffness relief have some precedent in TENS literature [Smith et al (1983), Taylor et al (1981), Dougherty (1979), Mannheimer et al (1984)], although the possibility of inaccuracy is inherent. However, Altman (1986) suggests that chronic sufferers of osteoarthritis of the knee are capable of being accurate when recording the diurnal variations of their pain. They learn over time to accommodate their daily activities to their pain state. Subjects were asked to indicate when, over a 24 hour period, their pain and stiffness levels, produced by the TENS trial, returned to "normal".

6.2.4 Objective Measurement

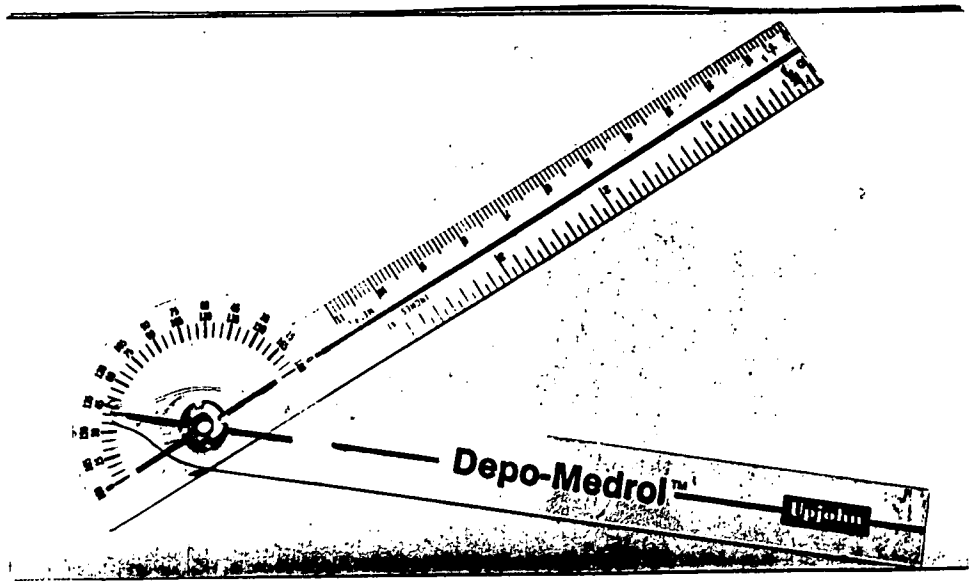
The objective measurements chosen for this study were those relating to changes in knee joint function after TENS application (Twomey 1979). The measurements would reflect local changes that may be caused by alteration in blood supply, changes in synovial activity, decreases in swelling and any local blocking activity that may occur with intrinsic opiate action.

6.2.4.1 Goniometer (FIG. 6B)

The use of the goniometer to measure changes in joint range was developed by Gifford (1914), during the First World War. Its uses have been considerably advanced since then [Rosen (1922), Moore (1949), Joshi, Singh, Kanta & Varma (1978)], although the standard goniometer remains a simple, easily used guide to knee joint movement. Twomey (1979) suggests that its measurement error is 1 degree. The use of a goniometer is supported in the TENS literature as a guide to objective joint improvement as a result of applying TENS (Medtronic 1984).

6.2.4.2 Tapemeasure

The use of a tapemeasure to measure changes in joint circumference has been well established [Apley (1978), Jacox (1977), Corrigan & Maitland (1986)]. It is an accurate and simple form of measuring physical changes in knee joint behaviour. This author allows an error of 0.5 cm when measuring around the joint line. To facilitate correct re-measuring after the test, ink lines were drawn on the skin at the top of the tape during the pre-test measurement. The tapemeasure was subsequently realigned with these marks for remeasuring.



Scale: 1 in 1.5

FIG. 6B The goniometer used in this study

CHAPTER 7

THE STUDY

7.1 Intervention (Study Factor)

This double blind experiment tested whether, when compared with High Rate TENS and a placebo, strong Burst Mode TENS produced significantly greater symptomatic change in chronically painful osteoarthritic knees. All TENS frequencies were applied to the same four acupuncture points for 30 minutes of constant, tolerable stimulation.

Any changes resulting from the application of Burst Mode TENS, High Rate TENS and placebo were measured by:

1. pain change on an Absolute Visual Analogue Scale (AVAS): readings taken immediately before and after the test,
2. stiffness change on an AVAS: readings taken as for 1.,
3. pain relief time (in hours) after the test,
4. stiffness relief time (in hours) after the test,
5. change in knee circumference: readings taken immediately before and after the TENS test,
6. change in knee joint range of movement: readings taken as for 5..

The subjects were tested in supine, with a small towel supporting the knee in approx. 15 degrees flexion. The four electrodes used were connected in parallel dual channels. All instructions were given, and measurements taken, by S.C., a typist, who was trained by this author in the protocol and measuring skills required for the study. S.C. was blinded to both the test design and the expected outcomes, and was not involved in the allocation of subjects into groups.

7.2 Subjects

7.2.1 The Reference Population

It was considered that all people with chronically painful osteoarthritic joints may benefit from this study. Pain relief gained from a successful TENS application may reduce medication, reduce time lost from work, prolong independence, postpone surgery and reduce visits to physiotherapy and allied health professionals.

7.2.2 The Source Population

The source population was limited by the small number of replies from those doctors contacted to supply subjects for this study. It comprised subjects from the following regions of Southern Tasmania. The referral sources are listed.

East Coast of Tasmania - 1 (referred by Royal Hobart Hospital RHH)
Tasman Peninsula - 2 (referred by Royal Hobart Hospital)
Northern Hobart suburbs - 19 (ref. RHH & Hobart General Practitioners.)
Eastern Shore of Hobart - 11 (ref. RHH & Hobart GPs)
Kingston - 12 (ref. Huonville Physio. Clinic & Huon GP)
Huon/Channel district - 31 (ref. Huonville Physiotherapy Clinic, Huon District Hospital, Cygnet Physio., Dover Annex, Huon GPs)

Total source population = 76.
All subjects suffered from painful, chronic osteoarthritis of the knee before April 1988. The methods of referral to the study are outlined in 7.2.3.2.

7.2.3 Obtaining the Study Sample

7.2.3.1 The study sample size was estimated using the results of a pilot study by this author in 1987 (lodged Q'ld University, Physiotherapy School), and information from the current TENS literature.

In the pilot study, High Rate TENS produced significantly greater and longer lasting pain relief than the placebo, with 20 subjects in each test group. Current literature suggests that Low Rate TENS (Burst Mode TENS) is expected to produce at least 1.5 X the length of pain relief created by High Rate TENS.

Given the constraints of this study (e.g. finance, time and availability of subjects), it was felt that 20 subjects in each group (60 overall) may be sufficient to confirm the hypothesis. Once the study was begun, it was realised that the small source population, obtained from the few replies to the proposed study, would have made a larger study impractical to conduct.

7.2.3.2 The method of obtaining the study sample was by:

1. compiling a register of all patients treated for osteoarthritis of the knee within the last six months, at the Royal Hobart Hospital (RHH) Physiotherapy Outpatient Clinic, the Huonville Physiotherapy Clinic, Huon District Hospital (HDH) Physiotherapy Department (including Dover Annex), Cygnet Physiotherapy and Kingston Physiotherapy,
2. compiling a register of subjects receiving private medical treatment for osteoarthritis of the knee. Letters were sent to
   i). all orthopaedic and rheumatology specialists in Southern Tasmania. Total = 7
   ii). General Practitioners in Hobart, Channel Area and Huon Valley, randomly selected from the telephone book. Total = 13

   TOTAL DOCTORS CONTACTED= 20

   NO. WHO RESPONDED = 7 General Practitioners (35% response rate)
   None of the specialists who were contacted for this study replied.

   iii). Letters were sent to all Senior Citizens Clubs in Hobart, and in the Channel and Huon Areas.
   TOTAL CONTACTED = 8
   NO. CLUBS WHICH RESPONDED = 2; netting 7 possible subjects.
   Those Senior Citizens who replied, were then followed up by contacting their General Practitioners, to assess their suitability for the test.

Table 7.1 The origin of the potential subjects in this study.

Place of treatment for Osteoarthritis of knee in last 6 months (January - June 1988)	No. subjects
Register of O.A. knee patients at Physiotherapy Outpatients Dept., Royal Hobart Hospital.	22
Huonville Physiotherapy Clinic	20
Huon District Hospital	9
Dover District Annex of H.D.H.	6
3 Huon District General Practitioners	7 (including 3 from 1 local Senior Citizen's Club)
4 Hobart General Practitioners	12 (including 4 from 1 local Senior Citizen's Club.)
TOTAL	76

Table 7.2 Particulars of potential subjects contacted for the trial.

Particulars		Male	Female
Died before the test } Would have been suitable		1	0
Refused to take part	Un-	0	1
Previous TENS experience	suit	1	1
No present knee pain	able	5	3
Unable to use AVAS		0	1
Too ill to participate		0	3
Number suitable to participate		23 +	37 = 60
TOTAL		30 +	46 = 76

7.2.3.3 The method of contacting possible subjects was as follows:

1. The doctor of any subject coming to the trial without referral was contacted. The subject's osteoarthritic knee diagnosis was confirmed, and permission was asked to involve the subject in the trial, should he/she be suitable.
2. The potential subject was contacted initially by telephone. The following items were addressed.
   i. The trial was explained, and it was ascertained whether the subject would be willing to participate.
   ii. It was ascertained whether the subject was willing to withold medication as outlined in 7.3.
   iii. The length of time and extent of O.A. affliction was discussed, whether the subject had present knee pain, whether one knee was more painful than the other, and if the patient could distinguish individual knee pain.
   iv. It was determined if the subject had previously experienced TENS therapy. If the subject or his doctor did not know, the subject's physiotherapist was contacted.
   v. A test time, the place and travel arrangements were then decided.
   vi. The subject was informed that pain relief should not be expected, and that he could withdraw at any time.

7.2.4 The Study Sample

Sixty suitable subjects (23 men and 37 women) were obtained from the 76 people contacted. The subjects in the study sample were 35 - 96 years of age, and had suffered from a chronically painful osteoarthritic knee for longer than six months. The possible sex frequency of osteoarthritis of the knee has been recognised, and the ratio of females to males found in this study (1.6 : 1) seems compatible with that indicated in the literature. The subjects and the independent measurer were blinded by not knowing into which group they were allocated, which TENS frequency was applied, or what were the expected outcomes.

These 60 subjects were randomly assigned by dice into three groups of 20. The dice was thrown by an independent observer (J.G., a typist) as each subject was enrolled into the study. Entry into the study occurred once all the criteria illustrated in 7.3 were fulfilled. J.G. filled out the test sheet (APPENDIX 2) with the subject's name, the group into which he/she was allocated, his doctor's name and the date and time of the test.

If the dice fell at 1 or 4, the subject was entered in Group 1. Group one was tested with High Rate TENS (80 Hz).

If the dice fell at 2 or 5, the subject was entered into Group 2. Group two was tested with Burst mode TENS (3Hz with trains of pulses of 80Hz).

If the dice fell at 3 or 6, the subject was entered into Group 3. Group 3 was the placebo application.

Each suitable subject's osteoarthritic knee diagnosis was confirmed with his doctor's records. In 51 cases the doctor had confirmed this diagnosis with X-ray and/or arthroscopy at some time previously. In 9 cases, the doctor suggested X-raying the subject, who he suspected had the disease, but had not yet had it verified by X-ray.

All but 4 of the subjects tested were able to independently attend the test. These four subjects were tested in Huon District Hospital.

The reasons for hospitalization were

1. three subjects with minor heart problems, and
2. one social admission due to recent bereavement.

Test venues were established in central places to accommodate local groups of subjects, so that travelling did not create a problem.

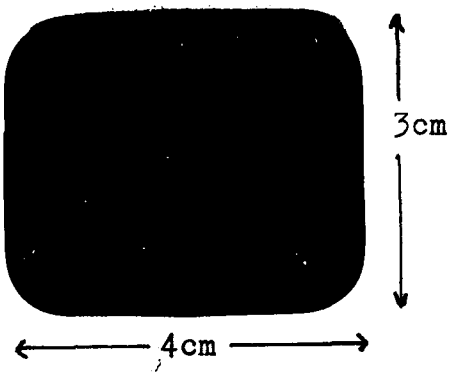
7.3 Inclusion Criteria for Subject Selection

1. Osteoarthritis was the only reason for knee pain, and hence referral to this study. The osteoarthritic state had been confirmed by the subject's doctor and had been diagnosed as chronic at least 6 months earlier. The criteria for chronic knee pain has been described in NH & MRC Report (1988) on "Management of Severe Pain". The subject must have present pain in his knee for the test to take place.
2. The subject's doctor considered him/her fit to participate in this study, and had given his consent for the subject to take part. No doctor refused permission for his patient to participate.
3. The subject had voluntarily withheld medication for the required period prior to, and after the test (See APPENDIX 3)
   a. NSAI medication for 48 hours before the testing period, and until the pain and stiffness levels returned to "normal" after the study.
   b. Analgaesics for 4 hours prior to the study, and until "normal" pain and stiffness levels returned.
   c. Muscle relaxants for 12 hours prior to the study, and until "normal" pain and stiffness levels returned.
4. The subject was not wearing a hearing aid or a pacemaker while experiencing TENS current (Eriksson, Schuler & Sjölund 1978).
5. The subject was prepared to sign a consent form (APPENDIX 1), which indicated that
   i. he/she agreed to participate in the trial of his/her own free will,
   ii. he/she was aware that pain and/ or stiffness relief was not guaranteed,
   iii. he/she had agreed to attend the test, and to be contacted 24 hours after the test, and
   iv. he/she could terminate his/her role in the trial at any time. All subjects completed the test.
6. The subject was able to complete visual analogue scales for pain and stiffness (APPENDIX 2). In the event that a subject be unable to complete the A.V.A.S. without help, the test would still take place, but the results would be excluded from the survey. All the subjects tested were able to fulfil the requirements of the test.
7. In the case of multijoint involvement with osteoarthritis, the subject must be able to distinguish the pain in his/her more painful knee.
8. The subject had no previous experience of TENS current.

7.4. Apparatus (Illustrated in FIG. 4B)

7.4.1 The Transcutaneous Electrical Nerve Stimulator used in this experiment was a Medtronic Neuromod Selectra, producing High Rate current of 80 Hz, and Burst Mode current, generated at 3 Hz, of trains of 7 pulses with an internal frequency of 80 Hz. It has an easy pushbutton method of increasing intensity, and a digital intensity display. It is powered by four replaceable AAA batteries. Although the life of batteries is greater than 60 hours performance, new batteries were used every 10 hours performance for maximum efficiency. The unit was independently checked during the trial for constant frequency output by Mr. Terry Reagan, of Medical Engineering Pty. Ltd., Hobart. The electrodes and active leads were similarly checked for performance.

7.4.2 Four carbon/rubber/silicon electrodes of 3 x 4 cm area (FIG. 7A) were used in a two channel configuration illustrated in FIG. 4D.



Scale: 1 in 1

FIG 7A Carbon-rubber Electrodes used for this experiment.

Para Gel was used as a contact medium under all electrodes, and Micropore paper tape was used to hold the electrodes in place.

A Stopwatch, test sheet and pen, couch, pillows, blanket, towel for knee support and a quiet room were test requirements.

7.4.3 The same goniometer was used for all objective range of movement measures of the knee (illustrated in FIG. 6B)

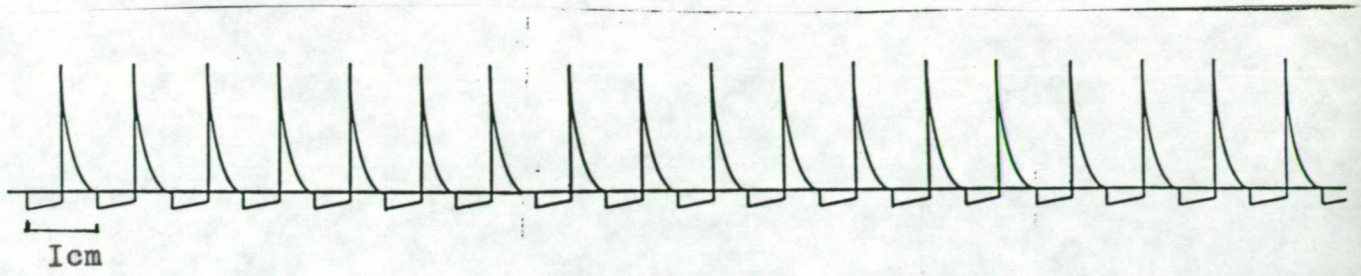
7.4.4 The same tape measure was used for all measurements of circumference of the knee around the joint line.

7.4.5 An appointments system was established, so that the test and follow-up times were well understood by experimenter, subject and measurer.

7.5 Frequencies of Tested Current

A Techtronics 422 oscilliscope was used to reproduce the currents produced in this trial by the Medtronics SELECTRA.

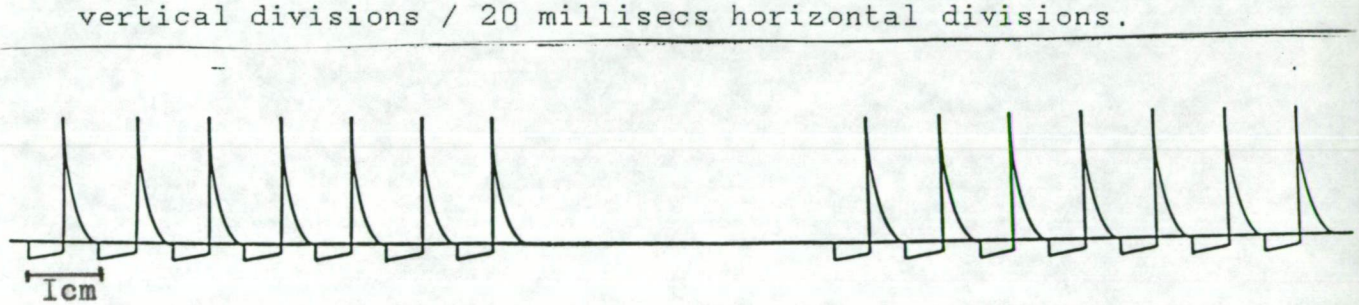
1. For group one, dual channel parallel currents of spike wave shape (fixed width 80 microsecs) at 80 Hz. (FIG. 7B) Oscilloscope parameters were 20 V vertical divisions, 20 millisecs horizontal divisions.



Scale: 1cm = 0.012 sec

FIG. 7B High Rate TENS (80 Hz) used in this experiment

2. For group two, dual channel parallel currents of spike wave shape fixed and applied as for group 1 were set at Burst Mode current generated at 3 Hz, comprising trains of 7 pulses of internal frequency of 80Hz (FIG. 7C). The oscilloscope was set at 5 V vertical divisions / 20 millisecs horizontal divisions.



Scale: 1cm = 0.012 sec

FIG. 7C Burst Mode TENS (3 Hz) used in this experiment

3. For group three, the placebo application was exactly the same as for the active TENS application (FIG. 7D). The subject did not experience any current flow. This was achieved by using dummy (inactive) leads supplied by Imbros Pty. Ltd., Hobart, on an active TENS machine. No current flow was seen on the oscilloscope.

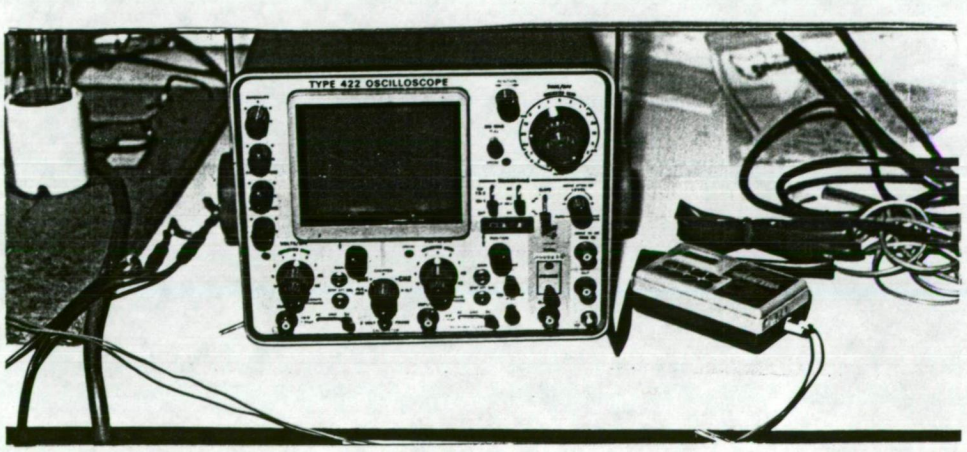


FIG. 7D
No current flow from placebo leads.
Scale: I in 4

7.6 Position for Experiment

The patient was supine with a rolled towel to support the painful knee 15 degrees from full extension (FIG. 7E). This position was proposed because:

1. the joint is not at extreme flexion or extension,
2. the vastus medialis, hamstrings and gastrocnemius are rendered inactive and therefore have no immediate bearing on joint pain [Hollinshead (1979), McConnell (1986)],
3. the joint capsule is not stressed (Levine 1979), and
4. it permits no 'rocking' of the knee, and no internal rotation of femur on tibia (Brunnstrom 1979). This would not place stress on the vascular, ligamentous, cartilagenous or neural structures of the knee.

FIG. 7E Subject in the position for the experiment

7.7 Stimulation Intensity

1. The intensity of the High Rate TENS application was set at just below patient pain threshold, producing a tolerable, tingling paraesthesia throughout the area of pain, without provoking a motor response.

2. The intensity of the Burst Mode TENS application was set at strong, tolerable level with visible muscle contraction.

3. The placebo group were told that a very high frequency current was being tested, and as the skin may not appreciate the current, no sensation may be felt under the electrodes. A light signal and the digital display indicated the pretended activity of the machine.

Stimulation levels were maintained by each subject by increasing the intensity regularly to maintain the perceived sensation. The subject was shown how to increase the intensity by pushing the increase buttons, and asked to increase the digital display by 3 points every 5 minutes (FIG. 7F i,ii). The subject was checked every 10 minutes during the test time, to ascertain whether the intensity was still satisfactory.

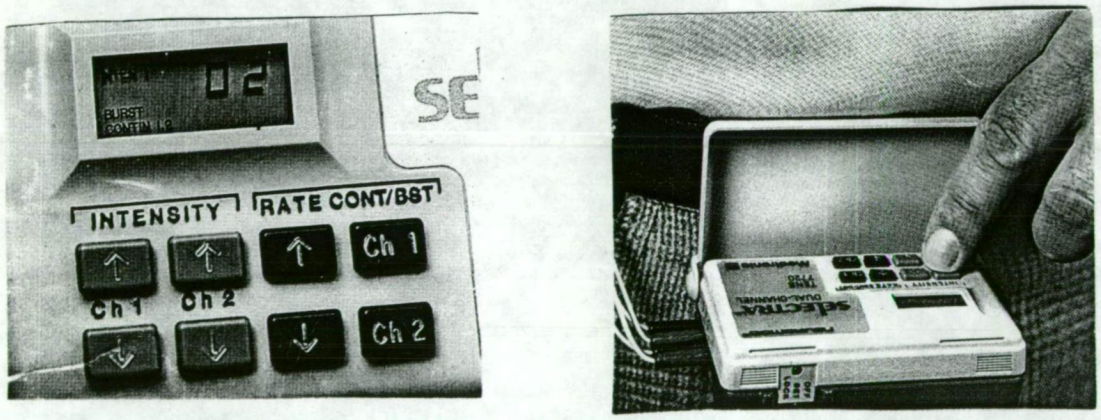


FIG. 7F(i), (ii) Push Button Intensity Increase

7.8 Electrode Placement

In all tests, the four electrodes were placed on acupuncture points Spleen 9, Spleen 10, Gall Bladder 33 and Urinary Bladder 40. The four points are academically described in CHAPTER 5 (Mann 1987).

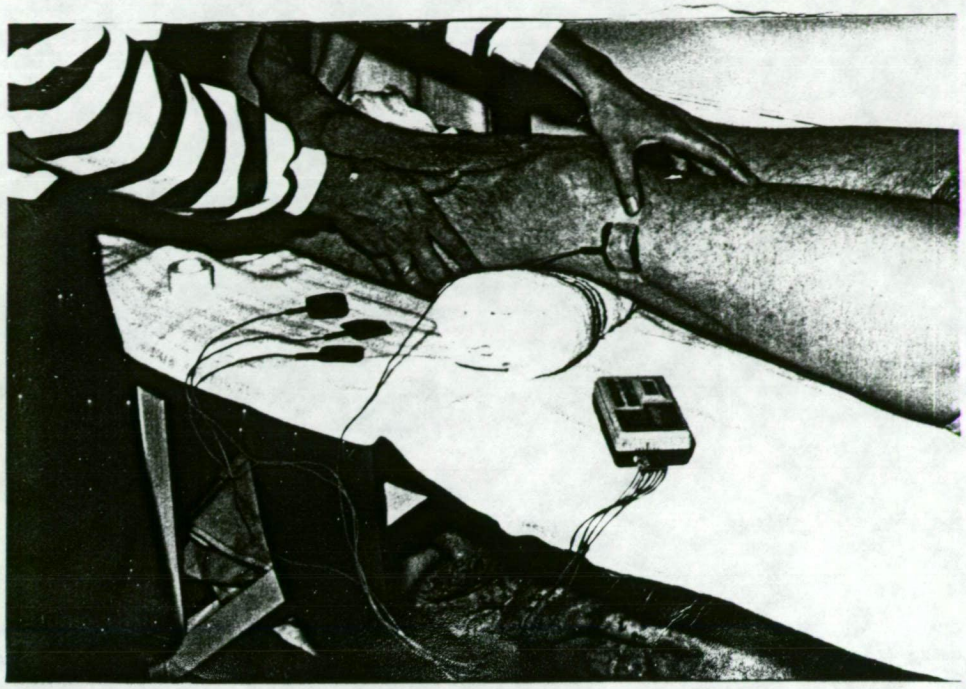


FIG. 7G Illustrating the electrode placement at Gall Bladder 33

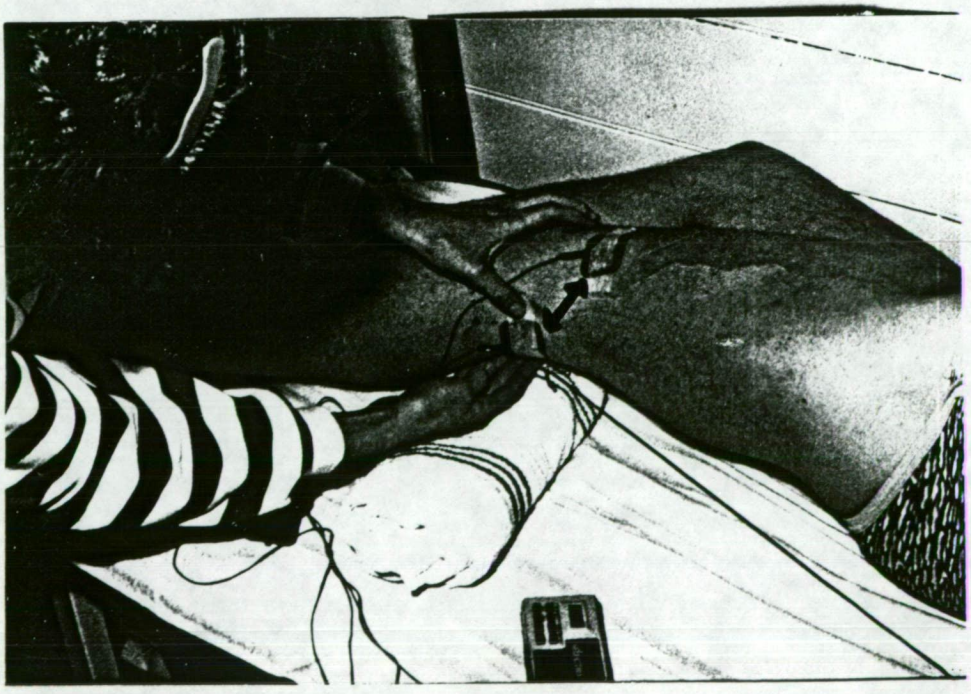


FIG. 7H Illustrating the electrode placement at Urinary Bladder 40

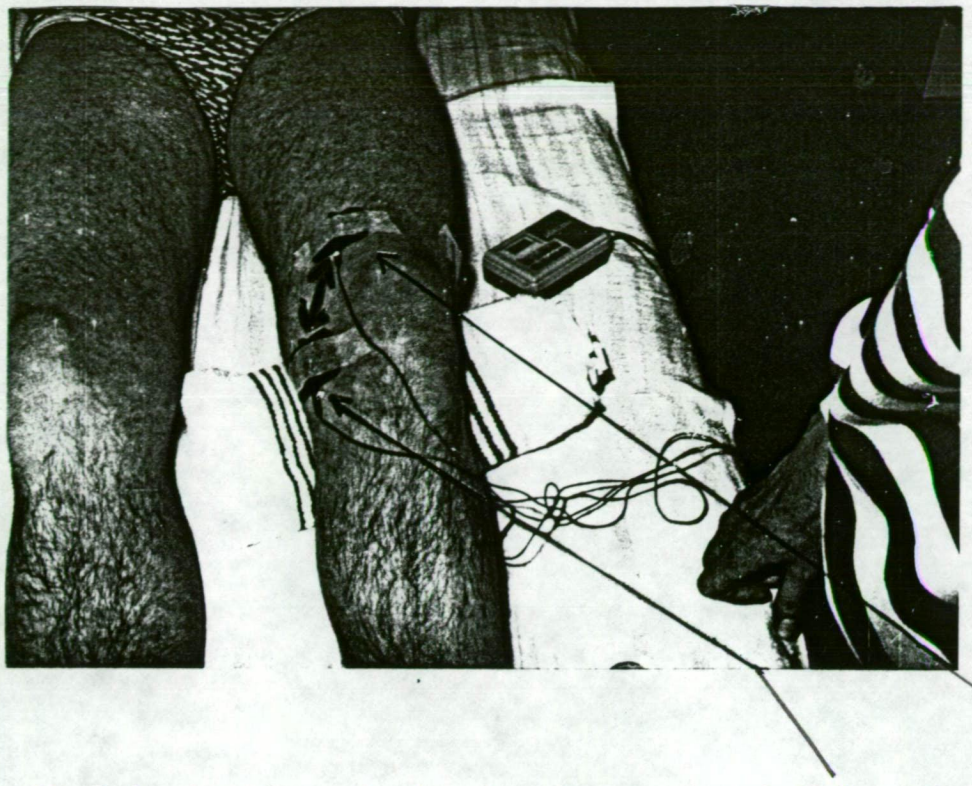


FIG. 7I Illustrating electrode placements at Spleen 9 & Spleen 10.

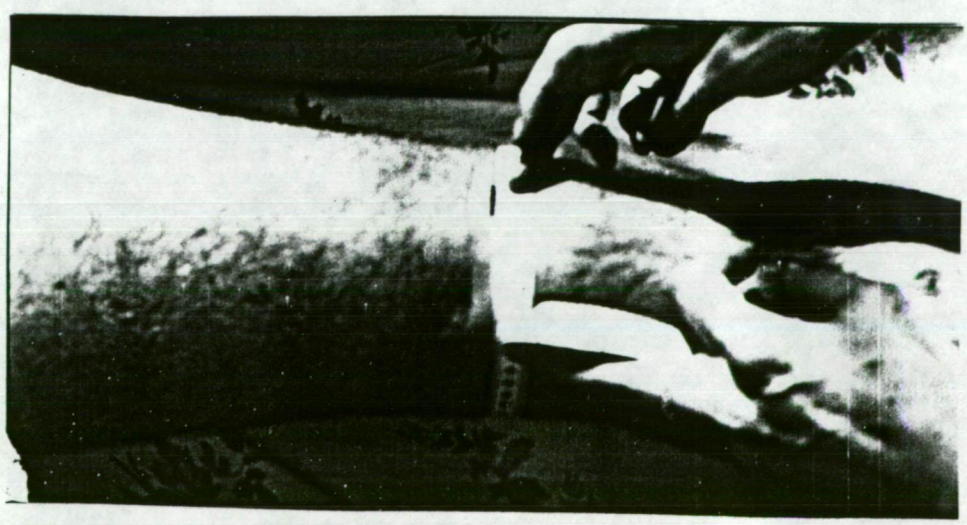
7.9 Outcome Factors

Measurements of knee circumference, range of knee joint movement, immediate pain and stiffness change, and length of pain and stiffness relief were made by one blinded independent observer (S.C.).

7.9.1 Objective measurements

1. The knee circumference was measured in centimeters (FIG. 7J) using the joint line as a guide. Marks were placed on the skin along the top of the tape at the pre-test measure as a guide to the post-test placement of the tape. Measurements of circumference were taken to the nearest 0.5 cm. The same tape measure was used for each test.

2. The comfortable Range of Movement was measured in degrees (FIG. 7K) before and after the test to the nearest degree. The same goniometer was used for all the tests. The femur and tibia were used as a guide for the arms of the goniometer.



Scale: I in 2

FIG. 7J Measuring the knee circumference.

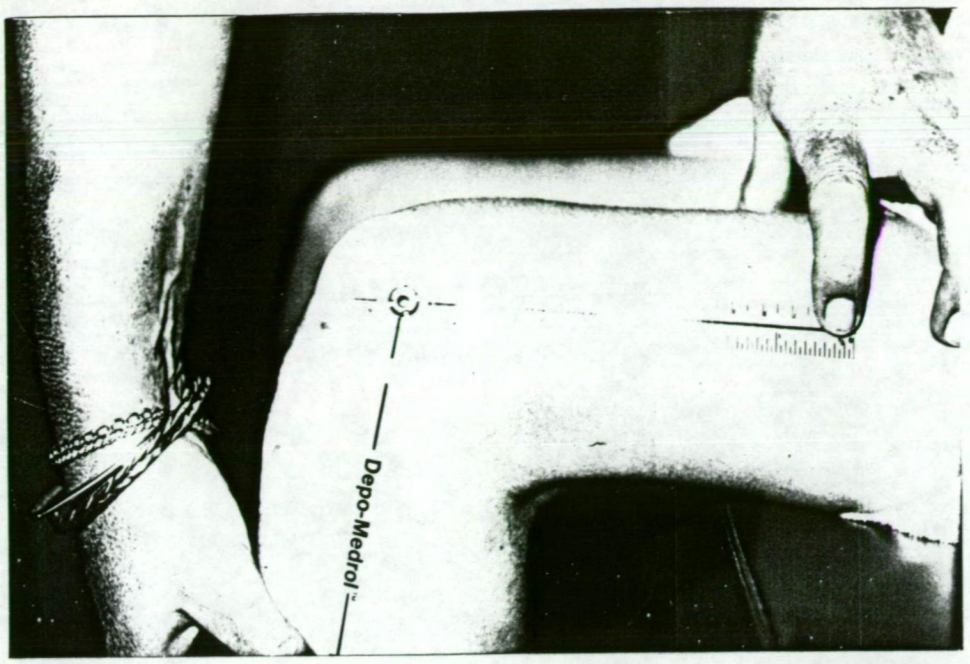


FIG. 7K Measuring the range of movement in this study.

7.9.2 Subjective Measurements.

1.. The change in pain state was indicated by the difference between pre-test and post-test Absolute Visual Analogue Scale measurements: ie the pain level was measured in centimeters, with an error of one decimal place (illustrated in APPENDIX 2).

2. The change in stiffness state was indicated by the change in Absolute Visual Analogue Scale readings as for pain.

3. The length of time the subject maintained his post-test pain and stiffness levels was reported in hours. It was found in all cases that subjects could be accurate as to times when their "normal" pain and stiffness levels returned. These measurements were taken to the nearest half hour of the return of normal pain.

7.9.3 The outcome factor measured whether strong Burst Mode TENS produced significantly greater sympomatic changes in painful osteoarthritic knees than did High rate TENS or the placebo.

7.10 Analysis of Data

The statistical analyses for this experiment were computed by Mr. K. Rayner, University of Tasmania Medical School, on SPSS - X Release 2.1 + Prime (SPSS User's Guide 1986).

Changes in pain levels, stiffness levels, circumference and range of movement as well as reports of pain and stiffness relief, were used to calculate any significant symptomatic change as a result of the TENS application. Means, standard deviation and pooled variance t-tests were calculated to indicate signifiance.

7.11 Possible Confounding Factors Which Were Controlled.

1. The time of day was a possible confounder. Pain appreciation changes diurnally, and the individual pain threshold becomes lower as the day progresses. All tests took place between 12 noon and 5 pm, to minimise any variation in pain appreciation. It was felt that all subjects would have been weightbearing for several hours by the test time, and this would have increased any mechanically created pain.

2. The season of the year also was a possible confounder with respect to pain threshold. Pain levels are greater in winter, so the tests were arranged to take place in July and August 1988, winter months in Hobart, Tasmania. The average temperature in Hobart during July 1988 was max. 13.7 degrees C.} av. temp.
   min. 6.1 degrees C.} 9.9 ^C.
   and the average for August 1988 was
   max. 14.0 degrees C.} av. temp.
   min. 6.1 degrees C.} 10.05^C.
   (Hobart Weather Bureau Monthly Report, July/ August 1988).

3. Sex bias which may have confounded results was addressed by randomly distributing the subjects. It was interesting to note that the suggested ratio of male and female subjects found in the literature was also found in this study.

4. The difference between acute and chronic pain of osteoarthritis was addressed by ensuring that all subjects had suffered from osteoarthritis of the knee for longer than 6 months.

5. Medication, a possible confounder of pain appreciation, was withheld prior to the test. The withdrawl of each of the possible types of medication was in accordance with the maximum half-life of the drugs within each class (Shane & Grant 1987). Medication was not restarted until 24 hours post-test. APPENDIX 3 illustates the half lives of all the arthritis drugs used by subjects in this trial.

6. Previous experience with TENS was eliminated as a confounder in the pre-test inclusion factors. Any subject who had experienced TENS current previously was not admitted to the trial.

7. Inability to use AVAS was considered an issue. However, no subjec had dificulty in using the AVAS in this study.

8. The blinded, independent measurer (S.C., a typist) was taught by K.A.G. to take the test measurements. Her skills were assessed by another physiotherapist, (J.C.), who was uninvolved with the trial, prior to any testing taking place. The line of the femur and tibia were taken as guides for placement of the goniometer arms, and ink marks were made at the top of the tapemeasure to facilitate remeasuring. The results were not communicated to the subject during or after the test and the measurer was unaware of which frequency was being administered to which group. This measurer (S.C.) also collected the times of return of "normal" pain and stiffness at 24 hours after the test.

7.12 Possible Confounding Factors Which Were Unable to be Controlled.

1. Age of the subject was a possible confounder. Although it may have been partly addressed by randomization, it was not possible to distribute age evenly in each group (Refer to section 8.9.1). The age of the subject may have altered his/her pain threshold and his expectation of the pain cycle. The age of the subject may have affected his exercise level and his diet as well.

2. The length of time the subject suffered from osteoarthritis was considered to be a possible confounder. The histograms seen in section 8.9.2 show that length of time of osteoarthritis diagnosis was unevenly distributed within each group. The length of time of diagnosis may have altered pain perception, pain expectation, limitations placed on the knee by the individual, and the expectation of a result.

3. Diet and exercise were possible confounders, when considering levels of Vit B3, necessary in daily food intake for Serotonin production. No effort was made to question subjects for their eating habits, as the experiment was not addressing diet or exercise in arthritic pain. However, to assess diet in relation to the subject's performance with TENS could be an area for future study.

4. Subjects were instructed to pursue their normal exercise habits after the test, as it was felt that "checking joint performance" may have occurred if some pain relief was the result of a TENS application (Mannheimer & Lampe 1984). It was not possible to enforce this, however, and undue exercise may have confounded the results.

7.13 Experimental Protocol

Send all participating subjects an information sheet some days prior to the test outlining the testing procedure (APPENDIX 1). At the test check that each subject understands the testing procedure, that he/she knows what is required and still agrees to participate.

1. Check that the subject tallies with the test sheet. Take remaining particulars of each subject while he/she is undressing. Take care not to imply that any relief of osteoarthritic sympoms will ensue.

2. While the subject is still standing, tell him, "This scale represents pain" (See APPENDIX 2). Ask the patient, "Will you mark on it the amount of pain you have in your knee at this moment? Notice one end of the scale corresponds to no pain, and the other end to pain as bad as it can possibly be".

3. Repeat this procedure using the stiffness scale on APPENDIX 2.

4. Position the subject lying comfortably and warmly with a support under the knee to rest it in 15 degrees flexion (See FIG. 7E).

5. Measure the knee circumference (FIG. 7J). Place a mark on either side of the knee at the top of the tape as a guide to later measure.

6. Measure the range of movement of the knee in full comfortable flexion and full extension (FIG. 7K).

7. Set the active TENS (Group 1 & 2) to the required pulse rate, checking that pulse shape is correct first.

8. Check that the placebo TENS leads are not passing any current.

9. Set the electrodes in place (See FIG. 7G, 7H, 7I).

10a. Explain to the Group 1 subjects that they will feel a "tolerable, tingling feeling throughout the area of the knee pain". Set the electrodes in place, adjust the intensity until the desired sensation is experienced. Ask the subject to maintain the sensation by increasing the intensity every 5 minutes by 2-3 mA (FIG. 7F(i,ii)).

10b. Explain to the Group 2 subjects that they will feel a "tolerable, pulsing feeling that will produce visible muscle contractions". Continue as for Group 1, and ask the subject to increase the intensity as for 10a. to maintain the sensation if required.

10c. Explain to the Group 3 subjects that they may not feel anything, as a very high frequency current, which the skin may not appreciate, is being tested for its ability to relieve pain. This instruction will be visually reinforced by the digital display on the unit. Ask these subjects to increase the intensity every five minutes throughout the test, by 2-3 mA, (as for 10a).

11. At the end of the test period, measure the knee circumference using the marks as tape guides. Also measure the range of movement in both full comfortable flexion and extension.

12. When this is completed, ask the patient to stand up and move around before marking his pain and stiffness levels on the visual analogue scales. Indicate on the scale which of the marks were pretest and which were post test.

13. Arrange to contact the subject in 24 hours, for the report of the times (if) when "normal" pain and stiffness returned. The subject must be reminded to continue with his normal activities over the next 24 hours, and not to abnormally rest or stress the knee.

14. Thank the subject for participating in this trial.

RESULTS

Abbreviations: AVAS - Absolute Visual Analogue Scale
cm - measured in centimeters
diff - difference between before and after test AVAS result measured in centimeters
$\bar{X}$ - mean
SD - Standard deviation
SE - Standard Error
B/T - Before Test
A/T - After Test
Group 1 - High Rate TENS
Group 2 - Burst Mode TENS
Group 3 - Placebo
Years O.A. - Length of time subject suffered from osteoarthritis.

TABLE 8.1 Particulars of subjects participating in the trial.

	Males	Females	Age			Years O.A.		
			$\bar{X}$	SD	SE	$\bar{X}$	SD	SE
Group 1	7	13	65.6	16.2	3.6	5.6	6.6	1.5
Group 2	8	12	65.7	16.5	3.7	9.9	10.5	2.3
Group 3	8	12	68.4	11.3	2.5	7.9	9.0	2.0
Total	23	37	66.5	14.6	1.89	7.8	8.9	1.1
	60							

When computing the results for this trial, the non-significance of the F-Value indicated that the t value and its significance be read from the Pooled Variance Estimate rather than the Separate Variance Estimate. This was an indication of the homogeneity of the three groups within the study sample.

TABLE 8.2.1 PAIN MEASUREMENT : Absolute Visual Analogue Scale in cm. (AVAS 1 from Data Sheet; App. 2)
Calculated on INDIVIDUAL DIFFERENCES in pain relief, not average diff.

	Before/Test			After/Test			Pain relief (cm B/T - cm A/T)		
	$\bar{X}$	SD	SE	$\bar{X}$	SD	SE	$\bar{X}$	SD	SE
Group 1	7.1	3.0	0.66	2.2	2.8	0.6	4.97	3.3	0.74
Group 2	5.9	2.3	0.51	1.5	1.8	0.4	4.43	1.8	0.40
Group 3	6.3	2.4	0.52	3.48	2.9	0.66	3.26	2.8	0.62

TABLE 8.2.2 Tests of significance (Pain Measurement)

i) Internal t Values (Comparing pain relief Before/Test with Pain Relief After/Test) 19 df.		
0.05 sig. level on 2 tails; Critical t value: t = 2.093		
	t value	2-tail prob.
Within Group 1	6.66	0.000
Within Group 2	11.00	0.000
Within Group 3	5.25	0.000

All TENS applications produce significant pain relief when comparing the before Test measurement with the after Test measurement within each group.

ii) Comparing Pain Relief between groups: 38 df				
0.05 sig. level on 2 tails: Critical t value: t = 2.021				
0.05 sig. level on 2 tails; Critical F value: F =~2.51				
	F Value	2-tail prob.	t Value (Pooled Variance Estimate)	2-tail prob.
Group 1 + Group 2.	3.43	0.01	0.64	0.52
Group 1 + Group 3.	1.41	0.46	1.56	0.12
Group 2 + Group 3.	2.43	0.06	1.32	0.19

There is no significant difference between the Burst Mode TENS, High Rate TENS and placebo TENS pain reducing ability.

TABLE 8.3.1 LENGTH OF PAIN RELIEF (Hours)

	$\bar{X}$	SD	SE
Group 1	15.8	9.9	2.2
Group 2	17.7	8.0	1.8
Group 3	10.2	10.2	2.3

TABLE 8.3.2 Test of Significance (Length of Pain Relief) with 38 df

0.05 sig. level on 2 tails; Critical T value: t = 2.021

0.05 sig. level on 2 tails; Critical F value: F =~ 2.51

	F value	2-tail prob.	t value (pooled variance estimate)	2-tail prob.
Group 1 + Group 2	1.53	0.36	-0.65	0.52
Group 1 + Group 3	1.06	0.90	1.77	0.085
Group 2 + Group 3	1.62	0.30	2.58	0.014

Burst Mode TENS only produced a significant length of pain relief when compared with the placebo.

TABLE 8.4.1 STIFFNESS MEASUREMENT : Absolute Visual Analogue Scale (AVAS2 in Appendix 2) (cm)
Calculated on INDIVIDUAL DIFFERENCES in Stiffness relief, not average.

	Before/ Test			After/Test			Stiffness Relief (B/T - A/T)		
	$\bar{X}$	SD	SE	$\bar{X}$	SD	SE	$\bar{X}$	SD	SE
Group 1	6.0	3.9	0.8	1.6	2.6	0.59	4.4	3.8	0.85
Group 2	5.7	3.6	0.8	2.1	2.8	0.64	3.5	3.3	0.74
Group 3	4.6	3.8	0.86	2.4	3.1	0.69	2.2	2.3	0.51

TABLE 8.4.2 Tests of Significance (Stiffness Measurement)

i) Internal t Value (19 df) (Comparing Before/Test with After/Test)
0.05 sig. level on 2 tails; Critical t value: t = 2.093

	t Value	2-tail prob.
Within Group 1	5.17	0.000
Within Group 2	4.76	0.000
Within Group 3	4.32	0.000

There is significant stiffness relief occuring in each of the TENS applications.

ii) Between Groups; Comparing Stiffness Relief between groups (38df)

0.05 sig. level on 2 tails: Critical t value: t = 2.021

0.05 sig. level on 2 tails: Critical F value: F =~ 2.51

	F value	2-tail prob.	t Value (Pooled Variance Estimate)	2-tail prob.
Group 1 + Group 2.	1.3	0.56	0.75	0.45
Group 1 + Group 3.	2.8	0.03	2.22	0.03
Group 2 + Group 3.	2.15	0.104	1.50	0.142

The only significant difference in stiffness relief is between High Rate TENS and the Placebo.

TABLE 8.5.1 LENGTH OF STIFFNESS RELIEF (Hours)

	$\bar{X}$	SD	SE
Group 1	16.35	10.6	2.38
Group 2	15.95	9.1	2.04
Group 3	7.05	9.2	2.04

TABLE 8.5.2 Tests of Significance (Length of Stiffness Relief) (38df)

0.05 sig. level on 2 tails; Critical t value: t = 2.021

0.05 sig. level on 2 tails; Critical F value: F =~ 2.51

	F value	2-tail prob.	t value (Pooled Variance estimate)	2-tail prob.
Group 1 + Group 2	1.37	0.50	0.13	0.89
Group 1 + Group 3	1.35	0.51	2.96	0.005
Group 2 + Group 3	1.01	0.98	3.08	0.004

There is a significant difference between the length of stiffness relief produced by Burst Mode TENS and the placebo, and between High Rate TENS and the placebo.

TABLE 8.6 Tests of Significance Between Length of Pain Relief and Length of Stiffness Relief in Each Group. (19df)

0.05 sig. level on 2 tails; Critical t value: t = 2.093

	$\bar{X}$ Diff	SD	SE	Correl-ations	2 tail prob.	t Value	2 tail prob
Group 1	-0.54	10.7	2.4	0.46	0.04	-0.22	0.82
Group 2	1.72	8.8	1.97	0.47	0.03	0.87	0.39
Group 3	3.1	8.9	1.9	0.57	0.01	1.56	0.13

There is no significant difference between lengths of pain relief and lengths of stiffness relief.

TABLE 8.7.1 CIRCUMFERENCE MEASUREMENT (cms)
Calculated on INDIVIDUAL DIFFERENCES, not average difference.

	Before /Test			After/ Test			Circumference Change		
	$\bar{X}$	SD	SE	$\bar{X}$	SD	SE	$\bar{X}$	SD	SE
Group 1	39.3	3.1	0.7	38.9	3.2	0.72	0.37	0.35	0.08
Group 2	40.6	4.5	1.0	39.8	4.5	1.02	0.77	0.75	0.16
Group 3	39.8	3.3	0.74	39.4	3.2	0.72	0.40	0.44	0.10

TABLE 8.7.2 Tests of Significance: Between Groups (38df)

0.05 sig. level on 2 tails; Critical t value: t = 2.021

0.05 sig. level on 2 tails; Critical F value: F ~= 2.51

	F Value	2 tailed Prob.	t Value (Pooled Variance Estimate)	2 tail prob.
Group 1 + Group 2	4.41	0.002	-2.15	0.04
Group 1 + Group 3	1.56	0.34	-0.20	0.84
Group 2 + Group 3	2.83	0.03	1.92	0.06

Burst Mode TENS produced a significantly greater change in circumference than High Rate TENS. It may have also produced greater change in circumference than the placebo, but this did not reach statistical significance.

TABLE 8.8.1 CHANGE IN RANGE OF MOVEMENT (degrees)
Calculated on INDIVIDUAL DIFFERENCES, not average difference.

	Before/ Test			After / Test			Change in R.O.M.		
	$\bar{X}$	SD	SE	$\bar{X}$	SD	SE	$\bar{X}$	SD	SE
Group 1	103.4	10.2	2.65	110.3	9.6	2.72	6.9	7.9	1.77
Group 2	97.8	9.2	1.76	108.0	9.3	2.54	10.2	12.9	2.88
Group 3	105.9	9.7	1.45	108.9	7.4	1.97	2.9	6.4	1.43

TABLE 8.8.2 Tests of Significance: Between Groups (38 df)

0.05 sig. level on 2 tails; Critical t value: t = 2.021

0.0.5 sig. level on 2 tails; Critical F value: F =~ 2.51

	F Value	2 tailed prob.	t Value (Pooled Variance Estimate)	2 tail prob.
Group 1 + Group 2	2.64	0.04	-0.96	0.34
Group 1 + Group 3	1.52	0.36	1.73	0.093
Group 2 + Group 3	4.02	0.004	2.23	0.03

Burst Mode TENS produced a significantly greater change in Range of Movement than High Rate or the Placebo.

8.9 Distribution of Confounders

8.9.1 Age

Histograms produced by SPSS - X Prime 2.1 Release

```
Count     Midpoint     One symbol equals approx. 0.20 occurrences

 1        28.67        *****
 0        32.00
 2        35.33        **********
 0        38.67
 1        42.00        *****
 2        45.33        **********
 3        48.67        ***************
 2        52.00        **********
 4        55.33        ********************
 5        58.67        *************************
 2        62.00        **********
 5        65.33        *************************
 6        68.67        ******************************
 7        72.00        ************************************
 3        75.33        ***************
 7        78.67        ************************************
 2        82.00        **********
 3        85.33        ***************
 4        88.67        ********************
 0        92.00
 1        95.33        *****
                       0    +    2    +    4    +    6    +    8    +
                       Histogram Frequency
Valid Cases = 60
```

FIG. 8A Age histogram for ENTIRE STUDY SAMPLE

```
Count     Midpoint     One symbol equals approx. 0.10

 1          28         **********
 0          31
 0          34
 1          37         **********
 0          40
 0          43
 1          46         **********
 0          49
 2          52         ********************
 0          55
 1          58         **********
 0          61
 2          64         ********************
 3          67         ******************************
 1          70         **********
 2          73         ********************
 3          76         ******************************
 0          79
 0          82
 1          85         **********
 2          88         ********************
                       0    +    1    +    2    +    3    +    4
                       Histogram Frequency
Valid Cases = 20
```

FIG. 8B Age Histogram for Group 1 (High Rate TENS).

```
Count      Midpoint      One Symbol equals approx. 0.10 occurrences

 1           36          **********
 0           39
 1           42          **********
 1           45          **********
 1           48          **********
 0           51
 1           54          **********
 3           57          ******************************
 1           60          **********
 0           63
 2           66          ********************
 1           69          **********
 1           72          **********
 1           75          **********
 1           78          **********
 2           81          ********************
 0           84
 2           87          ********************
 0           90
 0           93
 1           96          **********
                         0    +    1    +    2    +    3    +    4    +
                         Histogram Frequency
Valid Cases = 20
```

FIG. 8C Age Histogram Group 2 (Burst Mode TENS)

```
Count      Midpoint      One symbol equals approx 0.10 occurrences

 1           46          **********
 1           48          **********
 0           50
 0           52
 1           56          **********
 1           58          **********
 1           60          **********
 1           62          **********
 1           64          **********
 0           66
 3           68          ******************************
 0           70
 1           72          **********
 3           74          ******************************
 1           76          **********
 0           78
 2           80          ********************
 1           82          **********
 1           84          **********
 1           86          **********
                         0    +    1    +    2    +    3    +    4    +
                         Histogram frequency
Valid Cases = 20
```

FIG. 8D Age Histogram Group 3 (Placebo Group).

8.9.2 Length of time subjects suffered from osteoarthritis of the knee

```
Count       Midpoint     One symbol equals approx. 0.40 occurrences
 6              0        ***************
13              2        *********************************
 9              4        ***********************
 9              6        ***********************
 1              8        ***
11             10        ****************************
 2             12        *****
 2             14        *****
 0             16
 0             18
 3             20        ********
 0             22
 0             24
 0             26
 0             28
 2             30        *****
 0             32
 0             34
 0             36
 0             38
 2             40        *****
                         0    +    4    +    8    +    12    +    16
                         Histogram Frequency
Valid Cases = 60
```

FIG. 8E Length of O.A. Histogram (Entire Study Sample)

```
Count       Midpoint     One Symbol equals approx. 0.20 Occurrences

 1             .0        *****
 8            1.5        ****************************************
 0            3.0
 4            4.5        ********************
 2            6.0        **********
 1            7.5        *****
 0            9.0
 3           10.5        ***************
 0           12.0
 0           13.5
 0           15.0
 0           16.5
 0           18.0
 0           19.5
 0           21.0
 0           22.5
 0           24.0
 0           25.5
 0           27.0
 0           28.5
 1           30.0        *****
                         0    +    2    +    4    +    6    +    8    +
                         Histogram Frequency
Valid Cases = 20
```

FIG. 8F Length of O.A. Histogram Group 1 (High Rate TENS)

```
Count      Midpoint      One symbol equals approx. 0.20 occurrences

 2           0           ********************
 3           2           ******************************
 4           4           ****************************************
 2           6           ********************
 0           8
 3          10           ******************************
 0          12
 2          14           ********************
 0          16
 0          18
 2          20           ********************
 0          22
 0          24
 0          26
 0          28
 1          30           **********
 0          32
 0          34
 0          36
 0          38
 1          40           **********
                         0    +    1    +    2    +    3    +    4    +
                         Histogram Frequency
Valid Cases = 20
```

FIG. 8G Length of O.A. Histogram Group 2 (Burst Mode TENS)

```
Count      Midpoint      One Symbol equals approx. 0.20 occurrences

 2           0           **********
 3           2           ***************
 4           4           ********************
 2           6           **********
 0           8
 6          10           ******************************
 1          12           *****
 0          14
 0          16
 0          18
 1          20           *****
 0          22
 0          24
 0          26
 0          28
 0          30
 0          32
 0          34
 0          36
 0          38
 1          40           *****
                         0    +    2    +    3    +    4    +    5    +
                         Histogram Frequency
Valid Cases = 20
```

FIG. 8H Length of O.A. Histograms Group 3 (Placebo Group)

DISCUSSION

9.1 Key Findings

9.1.1 In this study, strong Burst Mode TENS has been shown to produce significantly greater effects when compared with High Rate TENS, only on the following measurement:
the immediate post-test change in circumference measurement.

The results from this study do not support the hypothesis that strong Burst Mode TENS produces significantly greater change in pain, stiffness, and range of movement than that produced by High Rate TENS, when both are tested on painful osteoarthritic knees. One would be uncomfortable with failing to reject the null hypothesis.

9.1.2 In this study, strong Burst Mode TENS did not produce significantly greater effects than High Rate TENS on the following measurements:

1. the immediate pain relief felt after the TENS test,
2. the post-test length of pain relief,
3. the immediate stiffness relief felt after the TENS test,
4. the length of stiffness relief after the TENS test, and
5. the immediate change in range of movement measurement.

9.2 Comparing the Study Results with the Author's Expectations.

Given the results of this study, discussion must continue about the presently accepted actions of High Rate, strong Burst Mode (Low Rate) and placebo Transcutaneous Electrical Nerve Stimulation with respect to painful osteoarthritis of the knee.

From the literature and clinical observation, this author expected that strong Burst mode TENS would produce effects significantly greater than those produced by High Rate TENS or the placebo, in all measurements taken in this study. On the available double blind data for TENS applied to osteoarthritic knees, strong Burst Mode TENS was expected to reproduce the Low Rate TENS effect, by producing 1.5 - 2 times the length of pain relief produced by High Rate TENS, but neither creating more than 6 hours pain relief.

From this study, the major notable issues which invite comment were
1. that Strong Burst Mode TENS did not produce significantly greater symptomatic changes than High Rate TENS, except on joint circumference.
2. the High Rate TENS effect on immediate osteoarthritic stiffness,
2. the size of the placebo response, and
3. the lengths of pain and stiffness relief produced by both High and Burst Mode TENS.

The results from this study indicate areas of research into TENS action that have not yet been fully explained in the literature. For adequate discussion to occur, the hypothesis must be considered in each of its components.

9.2.1 The Immediate Pain Relieving Effects of TENS

There was no significant difference when comparing the strong Burst Mode, High Rate and placebo effect on decreasing immediate pain, measured by the Absolute Visual Analogue Scale. It would appear that both active TENS applications had no greater effect on chronic osteoarthritic pain than the placebo. Both High Rate and Burst Mode TENS relieved the average pre-test pain by more than 50%, (20% and 30% respectively, greater than the placebo), suggesting that both TENS applications had been successful [Salar et al (1981), Hansson & Ekblom (1983)]. The placebo TENS relieved the mean pre-test pain by 45%. It could be argued that with a larger study sample, High Rate and strong Burst Mode results may approach more significant levels. Randomly assigning the subjects into the groups was expected to adequately distribute important differences in both pre-test and post-test pain appreciation.

9.2.2 The Length of Pain Relief After the TENS test.

Only strong burst mode TENS produced a significant length of pain relief when compared with the placebo. However, the low frequency TENS result was not significant when compared with the High Rate TENS result, and it could be argued that greater numbers of subjects may have created a significant High Rate response. So different were both the active TENS results to those suggested in the literature, that this author felt that it indicated further discussion.

Both the High Rate TENS mean (15.8 hours) and the Burst Mode TENS mean (17.6 hours), from this intervention, are in excess of those suggested in the literature on TENS for osteoarthritic knees. Double blind studies by Mannheimer & Lampe (1984), Smith et al (1983), Taylor et al (1981), and Thurin et al (1980) indicate that High Rate TENS pain relief will last 1 - 3 hours, with Low frequency TENS pain relief lasting up to 6 hours after stimulation ceases.

One of the factors determining subject selection in this experiment was that all subjects suffered from present, chronic pain in their knees. It has been shown that chronic pain sufferers have lower than normal levels of CSF and blood stream intrinsic opiates by virtue of their constant pain [Sjölund, Eriksson & Terenius (1977), Terenius & Wahlstrom (1975), Terenius (1979)]. Chronic pain patients could be expected to have different autonomic function and behaviour patterns from experimental pain subjects. It must be noted that Von Knorring et al (1978) indicated that the expectation of pain relief was directly related to the length of time the subject had suffered from chronic pain. Not only was this study sample affected by the withdrawl of the usual medication, but all tests took place on a winter afternoon when endorphin levels could be expected to be at their lowest. All subjects were ambulating before the TENS test, and mechanical and neural osteoarthritic pain mechanisms would have been established by the test time.

9.2.2.1 High Rate TENS Action on Length of Pain Relief.

Pain relief from High Rate TENS is expected while the larger, fast acting afferent fibres are being stimulated sufficiently to override the pain fibres, while the pattern of pain recognition may be re-established up to 1 hour after disruption by TENS. A tolerable intensity is considered necessary for effective stimulation. Although Merskey (1974) indicated the enormous higher cortical input

of personality, expectation and conditioning in pain appreciation, one could reasonably expect that the mechanical pain of an osteoarthritic knee would recommence immediately weightbearing was resumed.

Earlier experiments reporting the use of High Rate TENS as a pain relieving agent may not have been conducted over a sufficient length of time at sufficient stimulation intensity using the most effective electrode sites, to establish pain relief by any mechanism other than segmental &/or pattern disturbing action. Experiments such as those by Burton & Maurer (1974), Melzack (1975), Thorsteinsson et al (1977, 1978) were conducted over less than 20 minutes on spinal segments and/or nerve roots. Because the immediacy of High Rate TENS action may have been evident, the full possibilities of its action may not have been considered. The experiments conducted by Mannheimer & Lampe (1984), Dougherty (1979), Thurin et al (1980) and Taylor et al (1981) were over longer periods (25 - 30) minutes, at comfortable intensity, over cutaneous nerves, trigger points, and acupuncture points. These studies reported significant pain relief lasting after the treatment period but not in the order of the results of this study. The tolerable intensity, used in this study, was probably higher than that used previously, and may have contributed to the longer pain relief.

9.2.2.2 Burst Mode TENS Action on Length of Pain Relief.

It was expected that Burst Mode TENS would produce longer pain relief than High Rate TENS. This was because of the known endorphin action occurring after 30 minutes stimulation of strong intensity sufficient to create muscle contraction. Although the onset of pain relief with this stimulation frequency is slow, the effect is gradual and peaks after 30 minutes steady stimulation (Andersson & Holmgren 1976). It was anticipated in this experiment that the length of pain relief could be maximised by placing the electrodes over acupuncture points and maintaining a just tolerable intensity which created strong rhythmic muscle contractions. However, the length of pain relief exceeded expectation, although it was not in the ratio predicted by the literature when compared to the High Rate TENS result.

Tseng et al (1976) suggest that B-endorphin may take up to 10 hours to be enzyme degraded after stimulation ceases. Hormones such as Adrenocortcotrophin and B-lipotropin are released by the brainstem centres, along with intrinsic opiates, as a result of stress. These substances may increase an initial endomorphin response, but as their half lives are shorter than endomorphins, they may have a decreasing effect over time (Wilkes et al 1980). Thus it may be that the stress of attending an appointment, the withdrawl of medication and subsequent increase in pain, and having to focus on the behaviour of a chronically painful knee may have precipitated the release of additional pain mediating agents.

Although very little is written, it seems possible that Burst Mode TENS may also have some affect on the Pain Gate system, either by a fatigue mechanism over time, the effect of the train of high frequency pulses, or by chemical agencies via a negative feedback loop. As Low frequency TENS is known to progressively elevate pain threshold, its effect on disrupting neural pattern mechanisms, altering higher brain pain appreciation through memory, and altering physical movement expectation must be considered. Tests comparing the effects of pure Low Rate TENS and Burst Mode TENS are indicated to clarify any possible differences in action that have not been noted to date.

9.2.2.3 Placebo TENS Result

The effect of placebo TENS on chronic pain is reported by Thorsteinsson et al (1977) as 33%. Assuming this is based on the mean pain relief, in this study the effect is 40%.

The reaons for this response may be a combination of factors:

1. the application of TENS to incurable chronically painful knees may have created an expectation in excess of the actual effect of the current.

2. being invited to participate in a trial of this nature may have created an expectation.

3. the placebo effect created by this experiment may have been enhanced by the use of the visual display on the Selectra TENS used for the experiment.

4. the design of the experiment - in which the subjects were encouraged to increase the intensity of the TENS every few minutes - may have mediated a pain reducing effect by giving the subject active control over his own pain relief.

5. the subject's age and the length of time he had suffered from osteoarthritis may have affected the result.

9.2.2.4 Factors that may have influenced both High Rate and Burst Mode TENS Pain Relief

1. Combinations of some of the controlling factors in this experiment may have augmented the experimental results. The factors which may have influenced the results are thought to be:
   i. the 30 minute experimental length,
   ii. the tolerable, constant stimulation intensity,
   iii. placing electrodes over positions which were common to acupuncture points, common painful spots, trigger points and cutaneous nerves around the knee,
   iv. the experimental design, where each subject was encouraged to increase the intensity to maintain the TENS sensation.

2. Other factors which may have influenced the result may be:
   v. the effect of the muscle contraction, blood flow alteration and increased joint lubrication due to the electrical stimulation.
   vi. error in subject reporting of pain relief.
   vii. that factors which possibly enhanced the placebo effect may have also enhanced the reporting of true pain relief.

9.2.3 Immediate Post-Test Stiffness Relief

The only TENS application to have any significant effect at the 0.05 level was High Rate when compared with the placebo. It could be argued, that with greater numbers, the Burst mode TENS result may have been significant. This author's experience as a clinical physiotherapist has shown that stiffness is a real and definitive problem to many chronic osteoarthritic sufferers. It was interesting that no subject in this study had any difficulty differentiating between stiffness and pain, and in reports of length of pain and stiffness relief, the answer was often diffferent. 8 subjects (4 in Group 1, 2 in group 2, 2 in group 3) had no problem with stiff knees, while 7 subjects had more stiffness than pain.

Given the means of the pre-test stiffness measurement, it must be suggested that by chance the placebo group was less stiff than either active group. The placebo group also had a greater mean pre-test range of movement, which would authenticate this suggestion. There is no precedent in the literature for measuring the effect of placebo TENS on stiffness. However, it must be considered that these factors may have affected the placebo response to the stiffness measurement in this study. Histograms of pre-test stiffness measured on the Absolute Visual Analogue Scale, and the objective pre-test Range of Movement are illustrated for each group in APPENDICES 5 and 6.

A discussion on causal factors of stiffness is relevant in this section. Although the concept of stiffness is an abstract one, the causal factors are more likely to be objective and mechanical. Muscle tone and strength, blood supply, concentration of toxins within the joint, joint swelling and ligament dysfunction are all factors which could be expected to affect the stiffness of the joint. It was interesting to note that there was no correlation on the overall Spearman Matrix between pain and stiffness measurement on the Absolute Visual Analogue Scale, although there was consistent correlation between the stiffness measurements, circumference measurements and changes in Range of Movement (See APPENDIX 7).

Significant stiffness reduction after High Rate TENS poses some questions. In its original concept, High Rate TENS affected only the spinal gating mechanism, although the possibility of endomorphin stimulation has already been discussed. Cognitive influence, personality and expectation cannot be expected to play a large part in a measurement that is inherently objective. The fact that there is no significant overall relationship between pain relief and stiffness relief cannot suggest that as pain reduces, so does stiffness. There must be biomechanical factors acting on the joint to cause objective changes which could lead to a subjective assessment of a decrease in stiffness.

9.2.4 Length of Stiffness Relief Lasting After the Test.

Both High Rate and Burst Mode TENS created significant lengths of stiffness relief when compared with the placebo. Given that the experimental methods were identical, this result becomes interesting when it has been seen that Burst Mode TENS did not create significant changes in stiffness immediately after the TENS test. This author was prepared to accept that the endorphin action of Burst Mode TENS, possibly creating some local joint change in blood supply and lubrication, may not have peaked immediately after the test, thus not creating maximum stiffness relief until some time later. It was expected, however, that the overall stiffness relief would last longer after Burst Mode TENS that after High Rate TENS. This was not the case, and barring experimental error, questions as to the action of High Rate TENS must be asked. Is there an objective local change in joint behaviour after the tolerable intensity High Rate TENS application, that is not demonstrated by the Pain Gate Theory, cognitive influences, pattern generation, or intrinsic opiate production? The use of acupuncture points for electrode placement may have stimulated local objective changes hitherto only associated with lower frequency currents, although the size of the electrodes would have negated a pure acupuncture response. Without biochemical measures, these questions can only be hypothetical. However, the conventional use of High Rate TENS should perhaps be reassessed.

The placebo response in the length of stiffness relief measurement in this intervention invites questions. Given that stiffness is not directly related to pain change, any endorphin production and changes in joint behaviour generated by personality and expectation cannot be expected to have lasting effects on a measure such as stiffness. Given that the mechanical components of osteoarthritic pain would be re-established within a short period of recommencing weightbearing, the mean length of placebo stiffness relief is higher than could be anticipated from an application that should have had only a resting effect on joint performance.

9.2.5 Circumference Change

Strong Burst Mode TENS was the only TENS application to produce significant changes in joint circumference immediately after the test. The effect was expected by this author, due to the low frequency nature of the current and the already documented objective changes to joint performance created by Low Frequency TENS current [Scott (1969), Garl & Cooper (1979), Wolf, Gersh & Kutner (1978)]. The change in joint circumference could be expected because of:

1. the strong constant rhythmic muscle contraction,
2. the pumping action on the pockets of swelling within the joint,
3. the alteration in blood supply, and
4. local vascular and synaptic endomorphin action.

Given the experimental design, it was not possible for the joint circumference to be measured any later than immediately post test. Had this been done, a trend in TENS action may have become more apparent. This author finds it intruiging that although strong Burst Mode TENS creates significant circumference change it does not create significant immediate stiffness reduction. Although this may have been experimental error, it suggests that stiffness may have other causes than simply fluid stasis within the joint.

Because of the supported, comfortable, rested postion of the knee during the test, it may have become less swollen simply by not using it for the length of the test time. The placebo effect would be expected to to be minimal on such an objective measure.

9.2.6 Change in Range of Movement

Strong Burst Mode TENS produced a significant change in Range of Movement when compared with the placebo, although with greater numbers, High Rate TENS may have also produced a significant result. The significant change in circumference with Burst Mode TENS supports speculation that a change in Range of Movement could be anticipated, as a decrease in fluid around the joint could be expected to create greater freedom of movement within the joint. The correlations between overall stiffness change, change of circumference and the change in range of movement supports the theory that stiffness is related more to the objective measures than it is to pain.

Given the significance of the immediate post-test stiffness result from High Rate TENS, it is not surprising that it also appears to affect immediate change in range of movement. Further testing of High Rate TENS action on increasing range of movement could be supported. Again, because of the rested position of the painful joint during the test, the placebo effect was expected to be minimal.

9.3 Biases That May Have Arisen In This Study.

9.3.1 Sampling Bias.

The study sample was drawn from a limited population area. Although 2 subjects lived on the Tasman Peninsula, and one on the East Coast of Tasmania, the rest came from the Northern and Eastern Hobart, Kingston, and Huon/Channel area. The number of doctors who replied to the study proposal limited the study sample. The subjects most easily obtained came from the Royal Hobart Hospital and the author's local physiotherapy connections. Such a limited population may not have yielded a totally representative sample of subjects with osteoarthritic knees, that may have been assembled by sampling from a wider population frame. (ie all of Tasmania). It was interesting to note that despite the limited method and geographical areas of sampling, the ratio of women to men was similar to that suggested in the literature, and the numbers of men in each group were similar.

The question of external validity must be addressed. Given the confines of this study and the lack of time, medical support, financial and clerical assistance, the identification of suficient suitable subjects was a daunting task. That they may not be representative of the wider population of osteoarthritics is possible. However, given the significance of the results obtained in this study, and the fact that the ratio of male and female osteoarthritics in this test was comparable with that of wider analytical studies, it is suggested that further study into the areas addressed in this work may be supported in a more widespread fashion.

9.3.2 Selection Bias.

All the subjects who fulfilled the study criteria were randomised into groups by an unrelated person (typist; JG). Only one potential subject was eliminated (by death); another was enrolled in his place. As a result of this protocol, it is felt that no selection bias occurred.

9.3.3 Measurement Bias.

Measurement bias was minimised by the following steps;

1. One independent measurer (S.C.) took all measurements. This measurer was blinded to which TENS frequency was being delivered to which subject. She did not appreciate any possible outcomes from the study.

2. Only chronic sufferers from osteoarthritis (6 months or more) were tested. This was deemed to be the minimum length of time taken before pain becomes chronic (NH + MRC Report 1988). This should have standardised the emotional component of pain (Werner 1980) which may have affected individual pain threshold.

3. A standard assessment form was used for all tests.

4. A standard test protocol was administered.

5. The same Transcutaneous Electrical Nerve Stimulator, electrodes, active and placebo leads, gel, test position, goniometer and tapemeasure were used for each test.

6. All test results were recorded by S.C.; both immediately post test and at 24 hours.

7. The statistical analyses were done by Mr. K. Rayner of the Dept. Community Medicine of Tas. Medical School, although the author of this study entered the data. Again, given the time and financial constraints of this study, this was unavoidable.

8. The accuracy of the measurer (S.C.) must be questioned. She has no clinical background training to take physiotherapeutic measurements. However, she was trained by the author (K.A.G.), and her skill was assessed prior to, and during the testing period by an independent physiotherapist (J.C.) who was experienced as a clinical instructor. It was felt that errors made by S.C. during the testing process would be standard throughout the three groups, thus randomising any possible measurement error.

9. There may have been errors in the subject reporting of lengths of pain and stiffness relief after the test. These factors have already been discussed in 9.2.2.3.

9.3.4 Confounding Bias.

Some measurable biases, which may have confounded this study, were eliminated or minimised by controlling the study framework. These factors were discussed in CHAPTER 7. Other biases, which may have confounded the results, could not be controlled by this author in this intervention.

1. Age may have been a counfounder. The variations in age of subjects was large, and the distribution is shown in histograms in 8.9.1.

2. The length of time the subject suffered from osteoarthritis may have biased his attitude to pain and pain relief. However, there was no correlation either overall, or in any individual group between the pain and stiffness measurements before or after the test and the length of time the subject had suffered from osteoarthritis. The distributions of this possible confounder are illustrated in 8.9.2.

   There was a significant correlation in the Spearman Correlation Matrix between age and the length of time the subject had suffered from osteoarthritis, both overall and in the Group tested with Burst Mode TENS. The groups tested with High Rate TENS and the placebo did not have any significant correlation between age and length of time of O.A. It must be suggested that this relationship may have affected the attitudes of the subjects participating in the study.

3. The author was known to 22 of the 60 candidates. As the author is a local Huon physiotherapist with a well known interest in chronic pain management, expectations may have been created in those subjects who were familiar with the author. However, it was hoped that randomly assigning the subjects into the trial would distribute this expectation across the three groups.

4. The personal attention received when participating in this trial may have enhanced wellbeing and consequently altered pain thresholds. Two phone calls to the subjects prior to the test, a followup contact by letter prior to the test, the knowledge that the subject's doctor was being contacted, or had suggested them for the trial, the test, and the phone call at 24 hours, may have enhanced expectation.

5. An expectation of a result, despite information to the contrary, (verbally at the time of initial contact, and in the written Test Information Sheet- APPENDIX 1) may have been created in all subjects. The disappointment in ongoing pain relief measures currently available for chronic osteoarthritis of the knee may have engendered expectation.

6. The amount of rest or exercise done by each subject after the test was an uncontrolled feature of this experiment. Each subject was asked to continue his/her "normal" daily routine, but as many of the subjects had journeyed some distance to participate in this test, more exercise than normal may have been created simply by attending the test venue. The desire to privately assess a less than normally painful and stiff joint cannot be discounted.

7. Inadequate diet may have confounded the results.

8. The invitation to be involved with a study of this nature may have created the desire to help. A comment heard often during the study was "It may not help me, but it may help someone else". This may have biased the subjective results. Again, randomisation could be expected to evenly distribute this trend.

9.4 Conclusions

9.4.1 This experiment indicated that strong Burst Mode TENS does not produce uniformly greater symptomatic changes than High Rate TENS or a placebo, when both are applied at a tolerable intensity for the same lengths of time to acupuncture points on painful osteoarthritic knees.

9.4.2 Interesting results were produced by strong Burst Mode and High Rate TENS with respect to lengths of pain and stiffness relief after the TENS test. Further study is indictated to establish:

1. High Rate TENS effect on immediate joint stiffness.
2. whether these responses from High and Burst Mode TENS endure past 24 hours, and
3. whether they can be reproduced with repeated stimulation over time.

It is suggested that a larger study sample is required for more definitive results.

9.4.3 The placebo response needs further definition with respect to:

1. biochemical changes created both centrally and locally.
2. allowing the subject to control and visualise his own pain relief.
3. the suggestive nature of placebo TENS, and whether it endures past 24 hours.

9.4.4 The protocol of this intervention may have affected the results. Further interventions are indicated to test whether this may have happened. Future experimentation is needed to investigate:

1. the efficacy of acupuncture points for electrode placements for High Rate TENS, and the practicalities of electrode size to produce such effects,

2. the exact relationship of the effects of strong Burst Mode TENS and pure Low Rate TENS on pain and movement of osteoarthritic joints,

3. the lengths of High Rate and Burst Mode TENS applications necessary for maximum and ongoing effect,

4. the effects produced by different stimulation intensities,

5. whether other TENS frequencies produce different effects,

6. the efficacy of TENS on other chronic pain conditions.

9.4.5 A picture of effective clinical application of TENS to chronic osteoarthritic joints may be constructed from this intervention. The current literature is mostly nonspecific about parameters for treating particular conditions (Wolf & Gersh 1985). The legal, ethical and social ramifications suggested by Mannheimer & Lampe (1984) and Mannheimer (1987), indicate the need for specificity and definition to occur quickly in the clinical useage of Transcutaneous Electrical Nerve Stimulation. Without this, the physiotherapy profession may find itself unable to manage the growing expectations of their clients and allied health professionals. Osteoarthritis is an ideal condition to investigate, as it is one of the few conditions were TENS alone is acceptable to create pain relief. Although Mannheimer (1987) states "TENS does not treat anything - it must be used as an adjunct to other treatments", the change in pain, stiffness, swelling and movement created by TENS to osteoarthritic knees may be sufficient to establish improved useage. As other treatments are largely unsuccesful for this condition, few ethical questions can be raised with respect to experimentation.

The advantage of home use of TENS set at specific frequencies, specific intensities, and applied at particular sites for set lengths of time needs to be assessed for its long term benefit, not only as an aid to decreasing medication but also to increasing joint function and improving quality of life. It is felt that TENS manufacturers, doctors, lawyers, osteoarthritis sufferers and physiotherapists would benefit from such knowledge, not only for specifying equipment requirements, and advancing appropriate TENS applications, but also by helping to define the medico-legal implications that widespead prescription of Transcutaneous Electrical Nerve Stimulators for pain relief have raised.

APPENDIX 1

INFORMATION SHEET FOR TEST SUBJECTS

1. This procedure is completely safe and your doctor has approved this test procedure for you.

2. The machine being used in this test is a low voltage alternating current device applied through four electrodes around the knee for 30 minutes. You may or may not feel a tingling feeling under the electrodes.

3. You may not experience any change in your usual pain or stiffness in your knee.

4. You should not have taken any anti-inflammatory drugs within the last 48 hours, and muscle relaxants within the last 12 hours, and any painkiller within the last 4 hours. This will be discussed with you over the telephone before the test.

5. To complete the test, you are asked to take a note of your pain and stiffness over the next 24 hours. You will be contacted at 24 hours after the test for your results. Please note when the pain and stiffness you have at the end of the test returns to what you normally experience.

6. If you have experienced this type of machine before, please tell the experimenter.

7. If you wish, you can withdraw from this test at any time.

AUTHORITY and CONSENT FORM (accepted by the Ethics Committee of the Royal Hobart Hospital, 8.6.88)

I,.....of.....
acknowledge that I have volunteered to participate in this experiment conducted by Miss K.A. Grimmer. I understand that the procedure is safe, may or may not provide pain relief and that I am able to withdraw from the study at any time. I am happy to comply with the conditions of this study.

Signed .....

Witnessed.....

APPENDIX 2

Test Record Sheet for TENS testing MMedSci 1988

Date.....
Grp 1 / 2 / 3

Name .....
Age .....
Sex .....
Telephone No. .....
Time of Test .....
Usual Medication .....
Doctor's Name .....
Length of Osteoarthritis Diagnosis.....
Joint circumference B/T.....cm A/T.....cm
Passive Range of Movement B/T..... A/T..... degrees

Pain as bad as you have ever had it

No pain

Stiffness as bad as you have ever had it

No stiffness

==========

Return of "normal" pain

Time.....

Return of "normal" stiffness

Time.....

==========

1cm = 1cm

APPENDIX 3

SERUM HALF LIFE FOR DRUGS USED BY SUBJECTS in this STUDY.

1. NONSTEROID ANTI-INFLAMMATORY DRUGS used in the management of Degenerative Joint Disease (Shane & Grant 1987)

	USUAL DOSE	HALF LIFE (Hours)
Salicylates		
Ecotrin (low dose)	325 mg	0.75 - 2 (High dose)
	975 mg	12 - 26
Trisilate	655 mg	12 - 26
Arthropan	870 mg/5ml	12 - 26
Aspirin	250 mg	0.25
Nonsalicylates		
Voltarin	25, 100 mg	1.1 - 1.8
Dolobid	250,500 mg	8 - 12.5
Indocid	75 mg	6 - 20
Orudis	50 mg	1 - 4
Naprosyn	500 mg	12 - 15
Feldene	10, 20 mg	35 - 45
Clinoril	200 mg	16 - 20
Tolectin	400 mg	1 - 6
Brufen	400 mg	2

2. MUSCLE RELAXANTS used in the management of osteoarthritis (Martindale 1982)

Serepax	5 - 12
Prednisolone	2.5 - 5

3. ANALGAESICS used by subjects in the test (Martindale 1982)

Panadeine	2
Panadol	1.5 - 2
Panamax Co	2
Norgesic	3 - 14
Paracetamol	1 - 3.5
Digesic	4 - 12

APPENDIX 4

RAW DATA FROM STUDY

Abbreviations:
AVAS - Absolute Visual Analogue Scale
cm - measured in centimeters
Diff - difference between Before and After Test AVAS result measured in centimeters
P.I. - Patient Identity
B/T - Before Test
A/T - After Test

TABLE 1 GROUP 1 SUBJECTS (High Rate TENS)

PAIN DATA

P.I.	Sex	Age	Years O.A.	Test Time(pm)	AVAS B/T(cm)	AVAS A/T (cm)	Diff	A/T pain relief (hours)
AH	F	85	2.0	3.30	7.8	1.3	6.5	7
GB	F	65	1.0	12.00	9.8	0.0	9.8	24
RC	F	58	2.0	4.00	8.8	0.1	8.7	24
FB	F	88	30.0	1.00	6.9	5.2	1.7	19
GV	M	53	5.0	5.00	4.1	3.0	1.1	0
RB	F	77	6.0	12.00	10.0	5.9	4.1	24
BY	M	47	5.0	5.00	7.4	1.5	5.9	5
SH	F	64	2.0	12.30	2.4	1.8	0.6	19
DS	F	68	8	3.00	3.2	0.1	3.1	3
TG	M	28	2.0	12.00	4.2	0.2	4.0	24
MW	F	66	5.0	12.45	10.0	7.4	2.6	2
GB	F	67	2.0	1.00	10.0	0.0	10.0	1
GP	F	77	2.0	1.30	5.6	0.0	5.6	24
EV	F	51	1.0	1.45	10.0	9.1	0.9	17
EM	F	88	10.0	1.15	10.0	0.0	10.0	3
SG	F	74	11.0	12.00	2.3	0.8	1.5	24
RC	M	36	4.0	12.30	8.5	4.5	4.0	24
CR	M	77	10.0	12.00	9.1	2.3	6.8	24
RN	M	71	1.0	3.30	2.5	0.0	2.5	24
KH	M	72	6.0	2.30	10.0	0.0	10.0	24

TABLE 2 GROUP 1 SUBJECTS (High Rate TENS)

STIFFNESS DATA

P.I.	AVAS (cm) B/T	AVAS (cm) A/T	Diff (cm)	A/T Stiffness relief (hours)
AH	1.3	0.0	1.3	24
GB	0.0	0.0	0.0	0
RC	3.2	0.7	2.5	24
FB	7.0	5.5	1.5	24
GV	0.0	0.0	0	0
RB	10.0	1.8	8.2	24
BY	8.0	0.0	8.0	0
SH	10.0	1.1	8.9	24
DS	2.8	1.5	1.3	16
TG	0.0	0.0	0.0	0
MW	10.0	9.0	1.0	2
GB	0.5	0.0	0.5	24
GP	5.7	0.0	5.7	24
EV	7.0	6.0	1.0	18
EM	10.0	5.4	4.6	3
SG	10.0	0.2	9.8	24
RC	8.7	1.0	7.7	24
CR	8.8	0.0	8.8	24
RN	10.0	0.0	10.0	24
KH	7.2	0.0	7.2	24

TABLE 3 GROUP 1 SUBJECTS (High Rate TENS)

KNEE CIRCUMFERENCE DATA (in Centimeters)

P.I.	Circumference B/T	Circumference A/T	Circum. Change (cm)
AH	38.5	38.0	0.5
GB	40.5	40.0	0.5
RC	37	36.5	0.5
FB	38.5	38.5	0.0
GV	36.0	36.0	0.0
RB	44.5	44.5	0.0
BY	40	40	0.0
SH	41	41	0.0
DS	38.5	37.5	1.0
TG	36.0	36.0	0.0
MW	48.5	48.5	0.0
GB	40.5	40.0	0.5
GP	37.5	36.5	1.0
EV	39.5	39.0	0.5
EM	41.5	40.5	1.0
SG	37.5	37.0	0.5
RC	35.5	35.0	0.5
CR	36.5	36.0	0.5
RN	37.5	37.5	0.0
KH	41.0	40.5	0.5

TABLE 4 GROUP 1 SUBJECTS (High Rate TENS)

RANGE OF MOVEMENT DATA (in degrees)

P.I.	Flex. B/T	Ext. B/T	Flex. A/T	Ext. A/T	Change
AH	85	180	75	180	+10
GB	80	180	77	180	+3
RC	75	180	55	180	+20
FB	65	180	55	180	+10
GV	40	170	35	170	+ 5
RB	150	165	135	165	+15
BY	60	180	53	180	+ 7
SH	78	180	65	180	+13
DS	45	180	45	180	0
TG	55	180	55	180	0
MW	105	175	95	175	+10
GB	70	180	65	180	+5
GP	60	180	55	180	+5
EV	70	170	70	170	0
EM	115	170	85	170	+30
SG	60	180	60	180	0
RC	65	180	65	180	0
CR	60	180	60	180	0
RN	65	180	65	180	0
KH	80	180	75	180	+5

TABLE 5 GROUP 2 SUBJECTS (Low Rate TENS)

PAIN DATA

P.I.	Sex	Age	Years O.A.	Test Time(pm)	AVAS B/T(cm)	AVAS A/T (cm)	Diff (cm)	A/T pain relief (hours)
JF	F	66	14	3.30	7.4	1.2	6.2	14
EH	F	60	10	4.30	5.3	2.0	3.3	24
JB	F	36	3	12.30	7.8	5.2	2.6	24
SS	M	46	2	5.00	4.2	0.0	4.2	10
TB	M	42	2	3.30	3.2	1.5	1.7	17
HG	M	82	14	3.00	7.1	0.0	7.1	24
EW	F	66	2	1.00	5.0	0.0	5.0	24
LH	F	57	0.5	1.30	2.5	0.0	2.5	19
UW	F	80	30	12.00	7.1	2.8	4.3	19
RC	M	54	3	5.00	7.3	0.0	7.3	24
JM	M	68	10	12.00	3.4	0.9	2.5	2
OF	F	77	4	3.00	7.5	0.0	7.5	24
BL	F	56	20	1.30	4.4	1.7	3.7	20
KT	M	72	20	1.30	3.4	0.0	3.4	20
EL	F	88	5	1.00	10.0	5.5	4.5	24
MC	F	75	5	2.00	8.2	4.6	3.6	1
AB	F	96	40	1.00	2.8	0.0	2.8	3
JH	F	49	3.5	1.00	6.4	1.1	5.3	13
GJ	M	88	10.0	1.00	5.6	0.0	5.6	24
CH	M	56	1.0	1.00	10.0	3.5	6.5	24

TABLE 6 GROUP 2 SUBJECTS (Low Rate TENS)

STIFFNESS DATA

P.I.	AVAS B/T	AVAS A/T	Diff (cm)	A/T stiffness relief(hours)
JF	8.6	6.2	2.4	14
EH	0.4	0.0	0.4	24
JB	10.0	0.0	10.0	24
SS	3.9	0.0	3.9	18
TB	7.4	4.8	2.6	17
HG	1.2	0.0	1.2	24
EW	4.9	0.0	4.9	10
LH	0.9	0.6	0.3	24
UW	9.7	7.7	2.0	9
RC	0.0	0.0	0.0	0
JM	5.5	0.8	4.7	4
OF	5.2	2.6	2.6	24
BL	9.6	7.7	1.9	20
KT	0.0	0.0	0.0	0
EL	7.4	7.4	0.0	24
MC	8.0	6.5	1.5	8.5
AB	10.0	0.0	10.0	2.5
JH	5.0	3.6	1.4	24
GJ	6.5	2.7	3.7	24
CH	10.0	0.0	10.0	24

TABLE 7 GROUP 2 SUBJECTS (Low Rate TENS)

KNEE CIRCUMFERENCE DATA (in Centimeters)

P.I.	Circumference B/T	Circumference A/T	Change
JF	41.5	40.5	1.0
EH	42.0	41.5	0.5
JB	38.5	38.0	0.5
SS	35.5	35.5	0.0
TB	39	36.5	2.5
HG	38	38	0.0
EW	37.5	37.0	0.5
LH	46	45	1.0
UW	39.0	38.5	0.5
RC	36.5	36.0	0.5
JM	43.5	43.5	0.0
OF	41.5	40.5	1.0
BL	56.0	56.0	0.0
KT	44.0	42.5	1.5
EL	37.5	37.0	0.5
MC	40.0	39.5	0.5
AB	36.5	37.0	0.5
JH	38.5	37.0	1.5
GJ	40.5	39.5	1.0
CH	41.5	39.0	2.5

TABLE 8 GROUP 2 SUBJECTS (Low Rate TENS)

RANGE OF MOVEMENT DATA (in Degrees)

P.I.	Flex. B/T	Ext. B/T	Flex A/T	Ext. A/T	Change
JF	80	160	75	170	+15
EH	75	175	52	175	+23
JB	95	180	43	180	+52
SS	30	180	23	180	+ 7
TB	60	165	57	165	+ 3
HG	80	180	75	180	+ 5
EW	65	180	62	180	+ 3
LH	90	180	61	180	+29
UW	65	170	59	170	+ 6
RC	70	180	70	180	0
JM	95	170	80	170	+15
OF	75	170	65	170	+10
BL	80	180	70	180	+10
KT	55	180	55	180	0
EL	65	170	60	170	+5
MC	100	170	90	170	+10
AB	70	180	70	180	0
JH	135	180	120	180	+15
GJ	65	170	65	170	0
CH	95	180	80	180	+15

TABLE 9 GROUP 3 SUBJECTS (Placebo Application)

PAIN DATA

P.I.	Sex	Age	Years O.A.	Test Time(pm)	AVAS B/T	AVAS A/T	Diff (cm)	A/T pain relief (hours)
MP	F	79	0.6	1.30	8.1	7.0	1.1	3
GB	F	68	0.6	12.30	4.6	1.3	3.3	24
VW	M	71	10	2.30	7.4	4.9	2.5	13
HG	M	57	3	12.00	6.0	0.6	5.4	1.5
DF	F	56	10	12.00	8.1	3.4	4.7	3
BO	F	59	5	2.30	8.5	5.7	2.8	3
BR	M	75	11	3.30	2.7	2.7	0.0	0
DK	F	48	3	2.00	4.9	0.0	4.9	24
DM	F	73	10	12.00	4.4	0.5	3.9	11
WC	F	68	10	2.30	1.7	0.0	1.7	24
RC	M	73	20	3.30	8.2	0.5	7.7	20
JE	M	86	40	2.00	7.8	4.1	3.7	24
VE	M	68	1	1.30	3.1	3.1	0.0	0
JD	F	73	1.5	1.30	4.3	2.1	2.2	24
AL	F	61	3	1.15	10.0	1.0	9.0	7
EB	F	81	5	1.30	8.8	8.8	0.0	0
OM	F	84	2	1.30	5.8	5.8	0.0	0
MJ	F	79	10	2.30	8.7	8.4	0.3	0
AC	M	63	3	3.00	5.3	1.6	3.7	2
GL	M	46	10	12.30	8.3	0.0	8.3	20

TABLE 10 GROUP 3 SUBJECTS (Placebo Application)

STIFFNESS DATA

P.I.	AVAS B/T	AVAS A/T	Diff (cm)	A/T stiffness relief(hours)
MP	0.2	0.2	0.0	0
GB	7.4	5.4	2.0	24
VW	7.8	4.9	2.9	7
HG	0.7	0.0	0.7	1.5
DP	3.6	0.0	3.6	7
BO	3.8	1.0	2.8	6.5
DK	0.0	0.0	0.0	0
BR	2.7	2.7	0.0	0
DM	8.2	2.3	0.0	24
WC	0.8	0.0	0.8	0
RC	4.5	0.1	4.4	20
JE	10.0	4.9	5.1	16
VE	0.2	0.0	0.2	0
JD	4.4	1.9	2.5	24
AL	10.0	10.0	0.0	0
EB	9.0	9.0	0.0	0
OM	0.0	0.0	0.0	0
MJ	1.1	0.0	1.1	0
AC	7.9	1.0	6.9	5
GL	10.0	5.0	5.0	6

TABLE 11 GROUP 3 SUBJECTS (Placebo Application)

KNEE CIRCUMFERENCE DATA (in Centimeters)

P.I.	Circumference B/T	Circumference A/T	Change
MP	38.5	38	0.5
GB	46.0	46.0	0.0
VW	43.0	42.0	1.0
GV	36.5	36.5	0.0
DP	38.5	38.0	0.5
BO	35.0	34.5	0.5
DK	39	38.5	0.5
BR	38.5	38.5	0.0
DM	36.5	35.5	1.0
WC	39.5	39.5	0.0
RC	39.0	38.5	0.5
JE	40.5	40.5	0.0
VE	38.0	38.0	0.0
JD	43.5	42.0	1.5
AL	45.5	45.0	0.5
EB	40.0	39.5	0.5
OM	34.5	34.5	0.0
MJ	44.5	44.0	0.5
AC	37.5	38.0	- 0.5
GL	43.0	43.0	0.0

TABLE 12 GROUP 3 SUBJECTS (Placebo Application)

RANGE OF MOVEMENT DATA (in degrees)

P.I.	Flex.B/T	Ext B/T	Flex A/T	Ext A/T	Change
MP	63	180	55	180	+6
GB	73	180	74	180	-1
VW	65	165	55	165	+10
GV	70	180	45	180	+25
DP	55	180	52	180	+3
BO	50	180	45	180	+5
DK	110	180	112	180	+2
BR	80	180	80	180	0
DM	75	180	70	180	+5
WC	50	180	52	180	-2
RC	60	170	62	170	-2
JE	85	175	80	175	+5
VE	55	180	55	180	0
JD	80	180	80	180	0
AL	70	175	75	175	-5
EB	85	180	80	180	5
OM	55	180	55	180	0
MJ	60	175	60	175	0
AC	65	180	65	180	0
GL	135	180	130	180	+5

APPENDIX 5

Before test Stiffness Histograms as produced by SPSS - X Release 2.1 Prime

Measured on AVAS in cms.
ENTIRE GROUP

Count	Midpoint	One symbol equals approx. 0.40 occurrences
9	.0	**********
3	.5	********
4	1.0	**********
1	1.5	***
0	2.0	
1	2.5	***
2	3.0	*****
1	3.5	***
2	4.0	*****
2	4.5	*****
3	5.0	********
2	5.5	*****
0	6.0	
1	6.5	***
3	7.0	********
3	7.5	********
5	8.0	*************
2	8.5	*****
2	9.0	*****
2	9.5	*****
12	10.0	**********
		0 + 1 + 4 + 8 + 12 +
Valid cases = 60		Histogram frequency

FIG. 1 Before test stiffness histograms for total group

GROUP 1

Count	Midpoint	One symbol equals approx 0.10 occurrences
3	.0	*******
1	.5	***
0	1.0	
1	1.5	***
0	2.0	
0	2.5	
2	3.0	*****
0	3.5	
0	4.0	
0	4.5	
0	5.0	
1	5.5	***
0	6.0	
0	6.5	
3	7.0	*******
0	7.5	
1	8.0	***
1	8.5	***
1	9.0	***
0	9.5	
6	10.0	**********
		0 + 1 + 2 + 3 +
Valid cases = 20		Histogram frequency

FIG. 2 Before test stiffness histograms Group 1 (High Rate TENS)

```
GROUP 2
  Count     Midpoint    One symbol equals approx. 0.1 occurrences
  2          .0         ********************
  1          .5         **********
  2         1.0         ********************
  0         1.5
  0         2.0
  0         2.5
  0         3.0
  0         3.5
  1         4.0         **********
  0         4.5
  3         5.0         ******************************
  1         5.5         **********
  0         6.0
  1         6.5         **********
  0         7.0
  2         7.5         ********************
  1         8.0         **********
  1         8.5         **********
  0         9.0
  2         9.5         ********************
  3        10.0         ******************************
                        0....+....1....+....2....+....3....+....4....
  Valid cases = 20      Histogram frequency
```

FIG. 3 Before test Stiffness Histogram Group 2 (Low Rate TENS)

```
GROUP 3
  Count  Midpoint      One symbol equals approx. 0.10 Occurrences
  4        0.0         ****************************************
  1        0.5         **********
  2        1.0         ********************
  0        1.5
  0        2.0
  1        2.5         **********
  0        3.0
  1        3.5         **********
  1        4.0         **********
  2        4.5         ********************
  0        5.0
  0        5.5
  0        6.0
  0        6.5
  0        7.0
  1        7.5         **********
  3        8.0         ******************************
  0        8.5
  1        9.0         **********
  0        9.5
  3       10.0         ******************************
                       0....+....1....+....2....+....3....+....4....+....
  Valid cases = 20     Histogram frequency
```

FIG. 4 Before Test Stiffness Histogram Group 3 (Placebo Group)

BEFORE TEST RANGE OF MOVEMENT HISTOGRAMS - Flexion/Extension measurements shown on Histograms produced by SPSS-X Release 2.1 Prime

```
TOTAL GROUP
FLEXION
Count   Midpoint      One Symbol equals approx. 0.40 occurrences
 1        30          ***
 0        36
 1        42          ***
 3        48          *********
 5        54          ************
 7        60          ******************
10        66          **************************
 7        72          ******************
12        78          ********************************
 3        84          *********
 1        90          ***
 3       102          *********
 2       108          *****
 1       114          ***
 0       120
 0       126
 0       132
 2       138          *****
 0       144
 1       150          ***
                      0....+....4....+....8....+....12...+....16...+...
Valid cases = 60      Histogram Frequency
```

FIG 5(i) Before test Flexion Range of Movement Histogram Entire Group

```
TOTAL GROUP
EXTENSION

Count Value           One Symbol equals approx. 1.00 Occurrence
 1      160           *
 3      165           ***
 9      170           *********
 0      175
47      180           ***********************************************

                     0....+....10...+....20...+....30...+....40...+....
Valid cases = 60     Histogram frequency
```

FIG 5(ii)
Before test Extension range of Movement Histogram Whole Group

GROUP 1
FLEXION

Count	Midpoint	One symbol equals approx. 0.10 occurrences
0	35	
1	41	**********
1	47	**********
1	53	**********
4	59	********** **********
3	65	**********
2	71	**********
2	77	**********
3	83	**********
0	89	
0	95	
0	101	
1	107	**********
1	113	**********
0	119	
0	125	
0	131	
0	137	
0	143	
1	147	**********
0	155	
		0....+....1....+....2....+....3....+....4....+..
Valid Cases = 20		Histogram Frequency

FIG 6(i) Before test Flexion Range of Movement Group 1 (High Rate TENS)

GROUP 1
EXTENSION

Count	Value	One symbol equals approx. 0.40 occurrences
1	165	***
3	170	********
1	175	***
15	180	**********
		0....+....4....+....8....+....12...+....16...+..
Valid Cases = 20		Histogram frequency

FIG 6(ii) Before test Extension Range of Movement Group 1 (High Rate TENS)

==========

GROUP 2
FLEXION

Count	Midpoint	One symbol equals approx. 0.10 occurrences
0	23	
1	29	**********
0	35	
0	41	
0	47	
1	53	**********
1	59	**********
4	65	********** **********
2	71	**********
2	77	**********
3	83	**********
1	89	**********
3	95	**********
1	101	**********
0	107	
0	113	
0	119	
0	125	
0	131	
1	137	**********
0	143	
		0....+....1....+....2....+....3....+....4....
Valid cases = 20		Histogram Frequency

FIG 7(i) Before Test Flexion Range of Movement Histogram
Group 2 (Low Rate TENS)

GROUP 2
EXTENSION

Count	Value	One Symbol equals approx. 0.40 Occurrences
1	160	***
1	165	***
5	170	**********
1	175	***
12	180	**********
		0.........4.........8.........12.....
Valid Cases = 20		Histogram Frequency

FIG 7(ii) Before Test Extension Range of Movement Histogram
Group 2 (Low Rate TENS)

==========

APPENDIX 6 (cont)
GROUP 3
FLEXION

Count	Midpoint	One symbol equals approx. 0.10 Occurrences
0	42	
0	47	
2	52	**********
3	57	**********
3	62	**********
2	67	**********
3	72	**********
1	77	**********
2	82	**********
2	87	**********
0	92	
0	97	
0	102	
0	107	
1	112	**********
0	117	
0	122	
0	127	
0	132	
1	137	**********
0	142	
		0....+....1....+....2....+....3....+....
Valid Cases = 20		Histogram frequency

FIG 8(i) Before test Flexion range of movement
Group 3 (Placebo Group)

GROUP 3
EXTENSION

Count	Value	One symbol equals approx. 0.40 Occurrences
1	165	***
1	170	***
2	175	*****
16	180	**********
		0....+....4....+....8....+....12...+....16...
Valid Cases = 20		Histogram frequency

FIG 8(ii) Before test Extension range of movement
Group 3 (Placebo Group)

==========

APPENDIX 7 Spearman Correlation Coefficients

Table I3 Group I

	SEX	AGE	YRSOA	AVASB	AVASA	PNREL	AVAS2B	AVAS2A	PNREL2	CIRCB	CIRCA	FLEXB	EXTB	FLEXA	EXTA	DIFF	CIRCCHNG
AGE	-.3916 N(20) SIG .044																
YRSOA	.0275 N(20) SIG .454	.4515 N(20) SIG .023															
AVASB/T Pain	-.1650 N(20) SIG .242	.1134 N(20) SIG .317	-.0242 N(20) SIG .460														
AVASA/T	-.0092 N(20) SIG .485	-.1944 N(20) SIG .206	.1724 N(20) SIG .234	.0085 N(20) SIG .355													
Pain relief PNREL (hours)	.2139 N(20) SIG .183	.0387 N(20) SIG .436	-.0085 N(20) SIG .355	-.1234 N(20) SIG .302	-.1806 N(20) SIG .223												
AVAS2B Stiff-ness	-.0369 N(20) SIG .439	.3021 N(20) SIG .098	.3485 N(20) SIG .066	.0317 N(20) SIG .447	.2399 N(20) SIG .154	.1633 N(20) SIG .246											
AVAS2A	-.5342 N(20) SIG .000	.1054 N(20) SIG .329	.2880 N(20) SIG .109	.2377 N(20) SIG .156	.5583 N(20) SIG .005	-.2397 N(20) SIG .154	.4580 N(20) SIG .021										
PNREL2 stiffness relief.	-.1649 N(20) SIG .244	.4720 N(20) SIG .018	.0736 N(20) SIG .379	-.0462 N(20) SIG .423	-.0527 N(20) SIG .413	.4222 N(20) SIG .032	.3502 N(20) SIG .065	.0100 N(20) SIG .483									
CIRCB Circumference measure	-.4929 N(20) SIG .014	.2995 N(20) SIG .100	.0789 N(20) SIG .370	.5287 N(20) SIG .008	-.0103 N(20) SIG .483	-.3109 N(20) SIG .085	.3676 N(20) SIG .055	.3989 N(20) SIG .041	-.0780 N(20) SIG .371								
CIRCA	-.4473 N(20) SIG .024	.2424 N(20) SIG .152	.0412 N(20) SIG .432	.4744 N(20) SIG .017	.0360 N(20) SIG .440	-.3060 N(20) SIG .095	.3910 N(20) SIG .044	.3941 N(20) SIG .043	-.0605 N(20) SIG .307	.9882 N(20) SIG .000							
Flex B/T R.O.M. measure	-.4572 N(20) SIG .021	.3289 N(20) SIG .078	-.1345 N(20) SIG .286	.6612 N(20) SIG .001	.0176 N(20) SIG .471	.0053 N(20) SIG .491	.3507 N(20) SIG .060	.3677 N(20) SIG .055	.2020 N(20) SIG .196	.7455 N(20) SIG .000	.7436 N(20) SIG .000						
Ext B/T	.1797 N(20) SIG .224	-.0476 N(20) SIG .421	-.1001 N(20) SIG .337	-.4518 N(20) SIG .023	-.4580 N(20) SIG .021	.3014 N(20) SIG .049	-.2314 N(20) SIG .163	-.5124 N(20) SIG .010	.3281 N(20) SIG .079	-.3741 N(20) SIG .052	-.3542 N(20) SIG .063	-.3469 N(20) SIG .067					
Flex A/T	-.3116 N(20) SIG .091	.2898 N(20) SIG .108	-.1616 N(20) SIG .248	.6569 N(20) SIG .001	.0473 N(20) SIG .422	.0900 N(20) SIG .353	.4142 N(20) SIG .035	.3009 N(20) SIG .099	.1419 N(20) SIG .275	.6738 N(20) SIG .001	.6555 N(20) SIG .001	.9005 N(20) SIG .000	-.3936 N(20) SIG .043				
Extn A/T	.1797 N(20) SIG .224	-.0476 N(20) SIG .421	-.1001 N(20) SIG .337	-.4518 N(20) SIG .023	-.4580 N(20) SIG .021	.3014 N(20) SIG .049	-.2314 N(20) SIG .163	-.5124 N(20) SIG .010	.3201 N(20) SIG .079	-.3741 N(20) SIG .052	-.3542 N(20) SIG .063	-.3469 N(20) SIG .067	1.0000 N(20) SIG .000	-.3936 N(20) SIG .043			
Diff	.0273 N(20) SIG .455	.2550 N(20) SIG .139	.0300 N(20) SIG .437	.5853 N(20) SIG .003	-.5924 N(20) SIG .003	.1160 N(20) SIG .313	-.2041 N(20) SIG .194	-.3854 N(20) SIG .047	.0598 N(20) SIG .401	.2042 N(20) SIG .194	.1301 N(20) SIG .292	.3614 N(20) SIG .059	.1925 N(20) SIG .208	.2779 N(20) SIG .118	.1925 N(20) SIG .208		
Circ-chng	-.3266 N(20) SIG .080	.3080 N(20) SIG .093	.0542 N(20) SIG .410	.1958 N(20) SIG .204	-.4867 N(20) SIG .015	.0530 N(20) SIG .412	-.1625 N(20) SIG .247	-.0262 N(20) SIG .456	.1685 N(20) SIG .239	-.0563 N(20) SIG .407	-.1845 N(20) SIG .210	.0136 N(20) SIG .477	.1510 N(20) SIG .263	.0388 N(20) SIG .435	.1510 N(20) SIG .263	.4780 N(20) SIG .017	
Flex-chng	-.4389 N(20) SIG .026	.3227 N(20) SIG .083	.1455 N(20) SIG .270	.3116 N(20) SIG .091	.0458 N(20) SIG .424	-.2784 N(20) SIG .117	.1930 N(20) SIG .208	.3070 N(20) SIG .094	.0797 N(20) SIG .369	.5535 N(20) SIG .006	.5709 N(20) SIG .004	.6361 N(20) SIG .001	-.3176 N(20) SIG .086	.2925 N(20) SIG .105	-.3176 N(20) SIG .086	.2499 N(20) SIG .144	-.1404 N(20) SIG .266

Spearman Correlation Coefficients

Table I4 Group 2

	SEX	AGE	YRSOA	AVASB	AVASA	PNREL	AVAS2B	AVAS2A	PNREL2	CIRCB	CIRCA	FLEXB	EXTB	FLEXA	EXTA	DIFF	CIRCCHNG
AGE	-.1360 N(20) SIG .361																
YRSOA	-.1511 N(20) SIG .362	.6204 N(20) SIG .002															
AVASB	-.1772 N(20) SIG .227	.0036 N(20) SIG .363	-.0896 N(20) SIG .354														
AVASA	-.3526 N(20) SIG .064	-.1665 N(20) SIG .241	.0063 N(20) SIG .489	.5548 N(20) SIG .006													
PNREL	.0650 N(20) SIG .393	.0423 N(20) SIG .430	-.1807 N(20) SIG .223	.4131 N(20) SIG .035	.0000 N(20) SIG .500												
AVAS2B	-.3104 N(20) SIG .091	.0849 N(20) SIG .361	.1919 N(20) SIG .209	.3161 N(20) SIG .087	.5886 N(20) SIG .003	-.1870 N(20) SIG .215											
AVAS2A	-.2743 N(20) SIG .121	.0185 N(20) SIG .469	.2527 N(20) SIG .141	-.0060 N(20) SIG .490	.2469 N(20) SIG .147	-.4482 N(20) SIG .024	.3460 N(20) SIG .068										
PNREL2	-.1856 N(20) SIG .217	-.1058 N(20) SIG .329	-.3391 N(20) SIG .072	.3154 N(20) SIG .088	.2232 N(20) SIG .172	.4870 N(20) SIG .015	.0537 N(20) SIG .411	-.0691 N(20) SIG .386									
CIRCB	-.0710 N(20) SIG .383	-.0159 N(20) SIG .474	.1610 N(20) SIG .249	-.1774 N(20) SIG .227	.1218 N(20) SIG .305	-.0368 N(20) SIG .439	-.0276 N(20) SIG .454	.4103 N(20) SIG .036	.1270 N(20) SIG .297								
CIRCA	-.1864 N(20) SIG .216	.1408 N(20) SIG .249	.2841 N(20) SIG .112	-.1091 N(20) SIG .324	.0854 N(20) SIG .360	-.0087 N(20) SIG .485	-.0420 N(20) SIG .430	.3599 N(20) SIG .060	.1266 N(20) SIG .297	.9505 N(20) SIG .000							
FLEXB	-.3032 N(20) SIG .097	-.1771 N(20) SIG .228	-.1088 N(20) SIG .324	.3261 N(20) SIG .080	.3479 N(20) SIG .066	-.1649 N(20) SIG .244	.2627 N(20) SIG .132	.2203 N(20) SIG .175	.2841 N(20) SIG .112	.3104 N(20) SIG .091	.3590 N(20) SIG .060						
EXTB	.0101 N(20) SIG .483	-.1759 N(20) SIG .229	-.2006 N(20) SIG .198	-.0652 N(20) SIG .392	-.1707 N(20) SIG .236	.2774 N(20) SIG .118	-.2023 N(20) SIG .196	-.6448 N(20) SIG .001	.1240 N(20) SIG .301	-.3008 N(20) SIG .099	-.2488 N(20) SIG .145	.0216 N(20) SIG .464					
FLEXA	.0000 N(20) SIG .500	.1966 N(20) SIG .203	.1181 N(20) SIG .310	.2517 N(20) SIG .142	.0063 N(20) SIG .489	-.2761 N(20) SIG .119	.1949 N(20) SIG .205	.3223 N(20) SIG .083	-.0799 N(20) SIG .369	.1043 N(20) SIG .331	.1376 N(20) SIG .281	.6722 N(20) SIG .001	-.0936 N(20) SIG .347				
EXTA	-.0305 N(20) SIG .449	-.1823 N(20) SIG .221	-.1708 N(20) SIG .236	-.0320 N(20) SIG .445	-.1683 N(20) SIG .239	.2654 N(20) SIG .129	-.1873 N(20) SIG .215	-.6399 N(20) SIG .001	.1168 N(20) SIG .312	-.2896 N(20) SIG .108	-.2273 N(20) SIG .168	.0457 N(20) SIG .424	.9858 N(20) SIG .000	-.0640 N(20) SIG .394			
DIFF	.1151 N(20) SIG .314	.2620 N(20) SIG .132	-.0128 N(20) SIG .479	.6171 N(20) SIG .002	-.2295 N(20) SIG .165	.4597 N(20) SIG .021	-.1538 N(20) SIG .259	-.1262 N(20) SIG .298	.1728 N(20) SIG .233	-.2957 N(20) SIG .103	-.2293 N(20) SIG .165	-.0068 N(20) SIG .409	.0339 N(20) SIG .444	.3541 N(20) SIG .063	.0794 N(20) SIG .370		
CIRCCHNG	.0916 N(20) SIG .350	-.2278 N(20) SIG .167	-.3806 N(20) SIG .049	.1433 N(20) SIG .273	.0824 N(20) SIG .365	.1649 N(20) SIG .244	-.0390 N(20) SIG .435	.1655 N(20) SIG .243	.2289 N(20) SIG .166	.3028 N(20) SIG .097	.0929 N(20) SIG .348	.0141 N(20) SIG .476	-.2359 N(20) SIG .158	.0156 N(20) SIG .474	-.2264 N(20) SIG .169	.1654 N(20) SIG .243	
EXCHNG	-.2764 N(20) SIG .119	-.4024 N(20) SIG .039	-.3820 N(20) SIG .048	.0990 N(20) SIG .339	.4059 N(20) SIG .038	.0091 N(20) SIG .485	.0699 N(20) SIG .385	.0032 N(20) SIG .495	.5244 N(20) SIG .009	.3309 N(20) SIG .077	.3313 N(20) SIG .077	.6149 N(20) SIG .002	.1744 N(20) SIG .231	-.0243 N(20) SIG .459	.1222 N(20) SIG .304	-.3388 N(20) SIG .072	-.0380 N(20) SIG .437

Table I5 Group 3

	SEX	AGE	YRSOA	AVASB	AVASA	PNREL	AVAS2B	AVAS2A	PNREL2	CIRCB	CIRCA	FLEXB	EXTB	FLEXA	EXTA	DIFF	CIRCCHNG
AGE	-.0710 N(20) SIG .383																
YRSOA	.3604 N(20) SIG .059	.1290 N(20) SIG .294															
AVASB	-.1239 N(20) SIG .301	-.0117 N(20) SIG .480	.1424 N(20) SIG .247														
AVASA	-.2230 N(20) SIG .172	.5392 N(20) SIG .007	-.1071 N(20) SIG .215	.3074 N(20) SIG .046													
PNREL	-.0900 N(20) SIG .353	-.2410 N(20) SIG .153	.1086 N(20) SIG .324	-.1951 N(20) SIG .205	-.4430 N(20) SIG .025												
AVAS2B	.2040 N(20) SIG .194	.0064 N(20) SIG .409	.3844 N(20) SIG .047	.3079 N(20) SIG .046	-.1185 N(20) SIG .309	.2837 N(20) SIG .113											
AVAS2A	.0814 N(20) SIG .366	.1234 N(20) SIG .302	.0967 N(20) SIG .343	.2823 N(20) SIG .114	-.0803 N(20) SIG .356	.2221 N(20) SIG .173	.8361 N(20) SIG .000										
PNREL2	.1209 N(20) SIG .306	-.0745 N(20) SIG .377	.1874 N(20) SIG .214	-.0857 N(20) SIG .360	-.0589 N(20) SIG .403	.5521 N(20) SIG .006	.4821 N(20) SIG .016	.2985 N(20) SIG .101									
CIRCB	-.0710 N(20) SIG .383	.0280 N(20) SIG .453	.0311 N(20) SIG .448	.1946 N(20) SIG .206	-.1102 N(20) SIG .322	.4471 N(20) SIG .024	.4326 N(20) SIG .028	.4853 N(20) SIG .015	.1232 N(20) SIG .302								
CIRCA	.0089 N(20) SIG .485	.0216 N(20) SIG .464	.0399 N(20) SIG .434	.1509 N(20) SIG .263	-.1391 N(20) SIG .279	.3713 N(20) SIG .053	.4225 N(20) SIG .032	.4912 N(20) SIG .014	.0570 N(20) SIG .406	.9856 N(20) SIG .000							
FLEXB	.1777 N(20) SIG .227	.0087 N(20) SIG .485	.0976 N(20) SIG .341	.0128 N(20) SIG .479	-.3009 N(20) SIG .099	.2975 N(20) SIG .101	.4599 N(20) SIG .021	.5811 N(20) SIG .004	.1669 N(20) SIG .241	.3659 N(20) SIG .056	.3562 N(20) SIG .062						
EXTB	-.0233 N(20) SIG .461	-.1096 N(20) SIG .323	-.3977 N(20) SIG .041	-.5045 N(20) SIG .012	-.0120 N(20) SIG .480	-.1368 N(20) SIG .283	-.3013 N(20) SIG .098	-.2174 N(20) SIG .179	-.1236 N(20) SIG .302	-.4025 N(20) SIG .039	-.3412 N(20) SIG .070	.0486 N(20) SIG .419					
FLEXA	.0803 N(20) SIG .368	.1076 N(20) SIG .326	.1231 N(20) SIG .303	.0076 N(20) SIG .487	-.3332 N(20) SIG .076	.3085 N(20) SIG .093	.4670 N(20) SIG .019	.5764 N(20) SIG .004	.0765 N(20) SIG .374	.4096 N(20) SIG .014	.4799 N(20) SIG .016	.8760 N(20) SIG .000	.0000 N(20) SIG .500				
EXTA	-.0233 N(20) SIG .461	-.1096 N(20) SIG .323	-.3977 N(20) SIG .041	-.5045 N(20) SIG .012	-.0120 N(20) SIG .480	-.1368 N(20) SIG .283	-.3013 N(20) SIG .098	-.2174 N(20) SIG .179	-.1236 N(20) SIG .302	-.4025 N(20) SIG .039	-.3412 N(20) SIG .070	.0486 N(20) SIG .419	1.0000 N(20) SIG .000	.0000 N(20) SIG .500			
DIFF	.2483 N(20) SIG .146	-.6198 N(20) SIG .002	.2934 N(20) SIG .105	.2276 N(20) SIG .167	-.6621 N(20) SIG .001	.4368 N(20) SIG .027	.4145 N(20) SIG .035	.2111 N(20) SIG .186	.3767 N(20) SIG .051	.0733 N(20) SIG .379	.0408 N(20) SIG .432	.3372 N(20) SIG .073	-.2794 N(20) SIG .116	.2298 N(20) SIG .165	-.2794 N(20) SIG .116		
CIRCCHNG	-.4378 N(20) SIG .027	.1209 N(20) SIG .306	.0634 N(20) SIG .395	.3025 N(20) SIG .097	.2812 N(20) SIG .115	.1102 N(20) SIG .322	.1086 N(20) SIG .324	.0719 N(20) SIG .302	.2489 N(20) SIG .145	.2157 N(20) SIG .181	.1038 N(20) SIG .332	.0187 N(20) SIG .469	-.5030 N(20) SIG .012	.0294 N(20) SIG .451	-.5030 N(20) SIG .012	-.0389 N(20) SIG .435	
FLEXCHNG	.2444 N(20) SIG .149	.1092 N(20) SIG .323	.0807 N(20) SIG .368	.2108 N(20) SIG .186	.4807 N(20) SIG .016	-.2402 N(20) SIG .154	.1241 N(20) SIG .301	.1176 N(20) SIG .311	.1867 N(20) SIG .215	-.3208 N(20) SIG .084	-.3009 N(20) SIG .099	.1235 N(20) SIG .302	.1280 N(20) SIG .295	-.2443 N(20) SIG .150	.1280 N(20) SIG .295	-.1160 N(20) SIG .313	.1009 N(20) SIG .336

APPENDIX 7 Spearman Correlation Coefficients

Table I6 Total Numbers (Combined All Groups)

	AGE	YRSOA	AVAS0	AVAS1	AVAS2A	PNREL	AVAS2B	AVAS2A	PNREL2	CIRCB	CIRCA	FLEXB	EXTB	FLEXA	EXTA	DIFF	CIRCCHNG	FLEXCHNG
AGE	1.0000 (60) SIG .000	.0926 (60) SIG .239	-.1438 (60) SIG .136	-.1046 (60) SIG .079	.0451 (60) SIG .366	-.0567 (60) SIG .334	-.2315 (60) SIG .038	-.1465 (60) SIG .132	-.1736 (60) SIG .092	-.1[illegible] (60) SIG .107	-.2048 (60) SIG .058	.0404 (60) SIG .380	-.0745 (60) SIG .286	.0306 (60) SIG .408	.1297 (60) SIG .162	-.2203 (60) SIG .045	-.1367 (60) SIG .149
YRSOA		.3748 (60) SIG .002	-.0839 (60) SIG .262	.0651 (60) SIG .311	-.0549 (60) SIG .203	.1246 (60) SIG .171	.1076 (60) SIG .207	-.0740 (60) SIG .287	.0983 (60) SIG .227	.1375 (60) SIG .147	-.0066 (60) SIG .480	-.2623 (60) SIG .021	.0452 (60) SIG .366	-.2536 (60) SIG .025	.0654 (60) SIG .310	-.0802 (60) SIG .271	-.0594 (60) SIG .326
AVAS0			-.0301 (60) SIG .410	-.0168 (60) SIG .449	.0220 (60) SIG .434	.2479 (60) SIG .028	.2214 (60) SIG .045	-.0740 (60) SIG .287	.0983 (60) SIG .227	.1375 (60) SIG .147	-.0066 (60) SIG .480	-.2623 (60) SIG .021	.0452 (60) SIG .366	-.2536 (60) SIG .025	.0654 (60) SIG .310	-.0802 (60) SIG .271	-.0594 (60) SIG .326
AVAS1				.3338 (60) SIG .005	-.2698 (60) SIG .019	.1191 (60) SIG .182	.5122 (60) SIG .000	.0589 (60) SIG .327	.0294 (60) SIG .412	.9757 (60) SIG .000	.4860 (60) SIG .000	.1371 (60) SIG .148	.1577 (60) SIG .114	-.1526 (60) SIG .122	-.0244 (60) SIG .427	.1994 (60) SIG .063	.0204 (60) SIG .438
AVAS2A																	
PNREL																	
AVAS2B																	
AVAS2A																	
PNREL2																	
CIRCB																	
CIRCA																	
FLEXB																	
EXTB																	
FLEXA																	
EXTA																	
DIFF																	
CIRCCHNG																	
FLEXCHNG																	
	SEX	AGE	YRSOA	AVAS0	AVAS1	PNREL	AVAS2B	AVAS2A	PNREL2	CIRCB	CIRCA	FLEXB	EXTB	FLEXA	EXTA	DIFF	CIRCCHNG

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