Pulsed Electromagnetic Field Therapy for Exercise-Induced Muscle Injury

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ABSTRACT: A randomised, double-blind, placebo-controlled, trial was performed to assess the efficacy of Pulsed Electromagnetic Field (PEMF) therapy in treating experimentally induced muscle injury and delayed onset muscle soreness. Healthy volunteers (n=50) were made to perform multiple eccentric contractions of their triceps sureii muscle and were then randomised to receive daily treatments with either an active device or a placebo device. Measures of pain and stiffness, along with calf muscle tenderness, range of ankle motion, lower limb volume and serum creatinine kinase were measured at baseline and daily thereafter. Results: Subjects in the treatment group were found to have less overall pain ($p<0.02$) and tenderness ($p<0.05$) than the placebo group, with the difference being greatest from day 4 to day 7 ($p<0.05$). Subjects in the treatment group were also found to have greater range of movement and less ankle stiffness although these differences were not statistically significant until day 7 ($p<0.05$). No significant differences could be found between groups with regard to leg volume and serum creatinine kinase. Conclusion: The results of this study suggest that pulsed electromagnetic fields reduce pain and stiffness and enhance recovery time when applied to muscles after eccentric exercise. These findings raise questions as to the optimal treatment characteristics of this type of therapy as well as to the underlying biological mechanisms.

INTRODUCTION

Exercise-induced muscle injury is one of the most common types of trauma associated with physical activity. This injury is believed to result from muscle fiber damage produced by the strain of eccentric contractions where a muscle lengthens under tension. The injury produced by eccentric exercise has been well described and is associated with inflammation, tissue necrosis and the release of muscle enzymes such as creatinine kinase (CK). (Brown et al., 1997); Gleeson et al., 1995); MacIntyre et al., 1995). While this injury is believed to result from a combination of oxidative and mechanical stress (Armstrong, 1984)) the actual mechanism underlying the injury is not fully understood. (Pyne, 1994) .

Clinically, eccentric exercise produces the sensation of delayed onset muscle soreness (DOMS) which is a sensation of discomfort occurring two to three days after exercise. DOMS has been reported to be most evident at the muscle/tendon junction initially, and then spreading throughout the muscle. Recovery after a single bout of injury is generally complete, and subject to training effects whereby repeated bouts of exercise produce a lessened response. While the muscle soreness after this type of exercise typically peaks at day two or three after the exercise, an elevation in CK may occur as much as four or five days later, (Newham et al., 1986) with the levels at the time of maximal soreness providing a qualitative marker of muscle damage. (Fridén et al., 1989)

There are many different treatment modalities currently used for DOMS. The clinical-trial literature suggests that many of these may be of doubtful efficacy. While the pain and force of contraction have been found to be improved by anti-inflammatory medication such as ibuprofen or naproxen sodium, (Lecomte et al., (1998) there has been a lack of effect reported with cryotherapy, (Paddon and Quigley, 1997) Low level laser therapy, (Craig et al., 1996a) homoeopathy, (Vickers et al.,1997) Transcutaneous electrical neuro-stimulation, (Craig et al., 1996b) ultrasound, (Hasson et al., 1990) and massage. (Stiller et al., 1992)

Pulsed Electromagnetic Field (PEMF) therapy involves the application of electromagnetic fields to various parts of the body to assist in healing. The most thoroughly researched therapeutic application for pulsed electromagnetic fields is in the treatment of bone healing. This application originated from the work of Bassett and Becker, (Bassett and Becker, 1962) who described asymmetric voltage waveforms from mechanically deformed live bone. The mechanisms by which this therapy works it is thought to be through an acceleration of extracellular matrix synthesis, (Aaron and Ciombor, 1993) however, this is not entirely understood. In addition to an established role for use in bone healing, the therapeutic use of pulsed electromagnetic fields appears to have wider applications. (Sisken and Walker, 1995) There is evidence to support the use of PEMF in treating a diverse range of conditions, ranging from the healing of chronic venous ulcers (Ieran et al. 1990; Stiller et al. 1992; Todd et al. 1991) to joint disease such as rotator cuff tendonitis (Binder et al., 1984) and osteoarthritis, (Trock et al,. 1993) as well as treating acute soft tissue injury such as ankle sprain. (Wilson, 1972) This study is an attempt to assess efficacy of using PEMF therapy device compared with that of placebo, on the treatment of muscle injury produced by eccentric exercise.

METHODS

STUDY DESIGN AND POWER CALCULATION

A randomised, placebo-controlled, double-blind, methodology was used with a parallel study design whereby all subjects were exposed to one hour of eccentric exercise and then randomised to receive treatment with either an active device or an identical placebo device. Initial calculations based on changes in leg volume after eccentric exercise suggested that 20 subjects in each arm of the trial would detect a difference of 10%, with a statistical power of 0.8 and a p value of 0.05. Written informed consent was obtained from all subjects and the project was approved by the Monash University Standing Committee on Human Ethics.

SUBJECTS AND INDUCTION

Healthy volunteers (n=50, 30 male and 20 female) aged 18 to 45 (average age 25.4), who were not taking routine analgesics or involved in routine exercise were recruited for the study. Subjects were paid a small fee for their participation. After providing informed consent subjects were made to perform multiple eccentric contractions of the triceps sureii muscle by walking backwards on a treadmill placed on a 15 degree, downhill incline for one hour. This protocol was adapted from Jones *et al.* (Jones et al., 1997)

INTERVENTION

Subjects were randomised into two groups. The experimental group received treatment with a Magnafield® Magnetic Energy Resonance Induction Therapy device Model MF998 (Magnacare Pty. Ltd, Nailsworth, S.A.). This device generates a multi-rhythm pulsating induction magnetic field of 50 Hz which is pulsed at frequencies of 0.5, 1, 2, and 4 Hz using an induction coil located in a mat that is placed approximately 30cm from the body part to be treated. This waveform produced is a negatively biased with 55% on and 45% off in each duty cycle. The voltage to the coil in 13 volts RMS with a current of 0.1 amp which generates greater than 10 Watts and produces a field density of >0.04mT at a distance of 30cm from the coil. This field is insufficient to induce perceptible action potentials in sensory nerves or motor units.

The treatment protocol consisted of two 20 minutes sessions spaced 20 minutes apart, immediately after using the initial treadmill session and then daily for one week thereafter. In addition, treatments were given over-night for the two nights after the treadmill session. During these overnight sessions the magnetic induction unit was placed on a timing circuit so that the machine was switched on and off every twenty minutes throughout the night.

The control group received similar treatments with a sham device that was identical in appearance to the real unit but was modified so as to produce no magnetic induction. As treatment with the device produces no sensations, it was possible to effectively blind both the subjects and the researchers to the group allocation.

OUTCOME MEASURES

SUBJECTIVE PAIN MEASURES

Subjects were given diaries in which they were instructed to make daily recordings of maximum pain and stiffness using a Visual Analogue Scale (VAS). (Price et al., 1983) In addition, the diary included a modified form of the McGill Pain Questionnaire (Melzak, 1975) as well use of simple analgesics, ability to sleep and perform morning activities.

MUSCLE TENDERNESS (AS MEASURED BY MECHANICAL PAIN THRESHOLD)

Muscle tenderness was assessed at baseline and daily afterwards using a pressure algometer (Activator Methods, Inc. Phoenix, AZ) using standard methodology. (Fischer, 1987; Jensen, 1990) Measurements were taken at baseline and then daily at 6 points on the calf. The results for each day were averaged to obtain an overall result.

RANGE OF MOTION (ANKLE STIFFNESS)

Range of ankle motion was measured at baseline before the treadmill session and daily afterwards using a goniometer. The angle created between the base of the first metatarsal, the medial malleolus and the tibia was measured during the extremes of dorsiflexion and plantar flexion. Markings were made on the ankle joint to ensure that subsequent recordings used identical landmarks. The range of motion was calculated by subtracting the angle recorded at the extreme of plantar flexion from the angle at extreme dorsiflexion.

LOWER LIMB VOLUME

Lower limb volume was assessed before the treadmill session and daily afterwards. This was achieved using a displacement method whereby the subject's leg was placed into a specially constructed water container and the mass of the displaced water was measured. The water temperature was maintained at 35 degrees Centigrade.

SERUM CREATININE KINASE

Blood samples were taken to assess serum creatinine kinase levels before the exercise and for four days afterwards.

STATISTICAL ANALYSIS

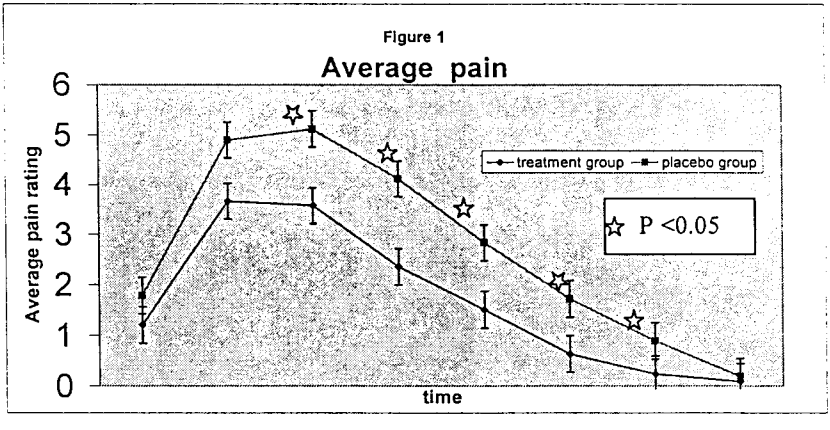
Statistical analysis was performed using SAS Version 6.12. (SAS, 1999) All outcome variables were checked for normality. As the variable CK found to follow a log-normal distribution, a logarithmic transformation was performed. Outcome variables were analyzed using a repeated measures analysis of variance adjusting for demographic variables (age & gender) and baseline values where appropriate.

RESULTS

EXCLUSIONS

Subjects were excluded from the analysis if they did not report any pain in the first six days after the treadmill session. Four subjects (three placebo and one active) were excluded in this way. The failure to report any pain after the exercise session was taken to indicate that these subjects did not fully comply with the treadmill protocol. It was found that during the treadmill sessions some subjects had difficulty balancing while walking backwards on the downhill-inclined treadmills. Consequently some subjects used the handrails to aid in balancing and it is possible that a few of these subjects also used the handrails to support their weight (although instructed not to). This would have the effect of greatly reducing the force of the eccentric contractions of their calf muscles and effectively preventing significant injury.

AVERAGE PAIN (FIGURE 1)

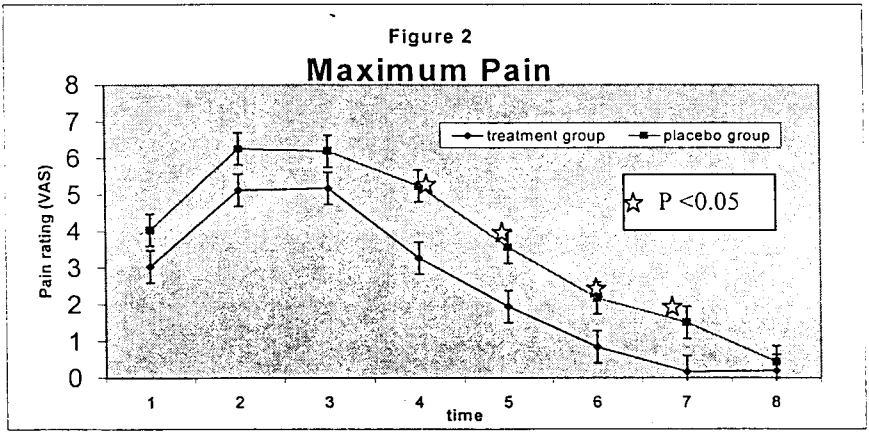


The daily pain rating was defined as an average of pain scores at four different time points throughout the day (morning, afternoon, evening & night). When daily pain measurements were averaged over the duration of the week, average pain was found to be normally distributed and as such was analyzed using an analysis of variance adjusting for age and gender. Subjects in the treatment group were found to have an average pain level lower than placebo group subjects (1.65 vs 2.76 p=0.02). To further investigate the differences between groups with regards to pain, a repeated measures analysis of variance was used with adjustments for age and gender. Subjects in the treatment group were once again found to have significantly lower levels of pain (1.66 vs 2.7 p=0.014). Pain was found to significantly decrease with time although this decrease was not consistent between groups, with the treatment group significantly less pain than the placebo group from day four to day seven, ($p<0.05$).

MAXIMUM PAIN SCORE (FIGURE 2)

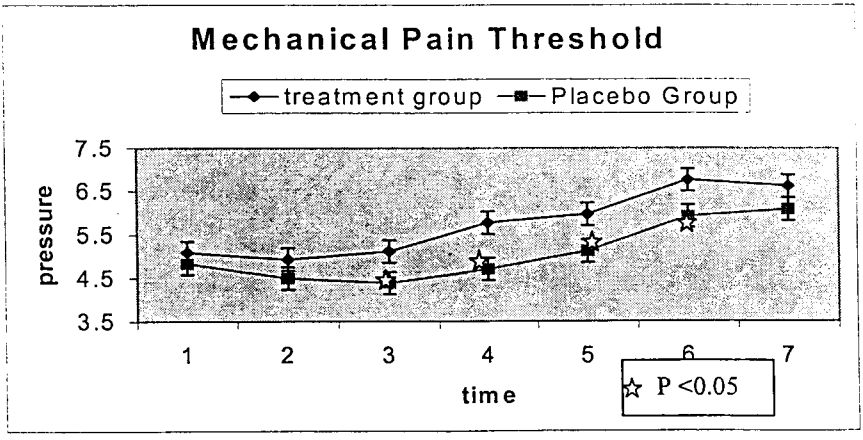
The maximum pain score recorded by subjects on a 100mm Visual Analogue Scale (VAS) asking subjects to score the most pain they had experienced in the past 24 hours. The maximum pain score was found to be significantly lower for the treatment group in comparison to the placebo group. (average soreness overall 2.50 vs 3.78 p=0.016). This result was confirmed in the repeated measures analysis (2.47 vs 3.67 p=0.013). As with the daily pain rating, the difference between groups with regards to maximal pain is most apparent on days four through to seven where there was a significant difference between groups ($p<0.05$). Age (p=0.05) was also found to effect maximal pain score.

Figure 2



MECHANICAL PAIN THRESHOLD (FIGURE 3)

Mechanical Pain Threshold (MPT) was defined as the average of six algometer readings taken at different sites over each subject's triceps sureii muscle. The treatment group was found to have a significantly higher average pressure score per leg in comparison to the placebo group (5.9 vs 5.2 p=0.05). Males were found to have a significantly higher score than female (6.1 vs 5.0 p=0.003), and tolerance to pressure was found to increase with time. The effect of time was found to be significantly different between groups with the treatment group subjects having significantly higher scores on day three through to day seven ($p<0.05$). Age and "which leg" (left or right) had no effect on pressure.



RANGE OF MOTION

Range of motion of the ankle joint was calculated for each subject by subtracting the angle of plantar flexion from the angle of dorsiflexion. Angle was found to be normally distributed and was thus analyzed using a repeated measures analysis of variance adjusting for angle at baseline, age, gender and which leg (left or right). Subjects in the placebo group were found to have less range of movement overall (55.7 vs 60.4) although this was not statistically significant ($p=0.10$). Time was found to have a significant effect on angle with all subjects having an increased range of movement as time progressed. This effect was found to be significantly different between groups ($p=0.005$) with subject in the treatment group having a significantly higher range of movement on day seven (65.7 vs 54.5 $p=0.0005$) in comparison to the placebo group.

ANKLE STIFFNESS

Stiffness was treated in identical fashion to maximal pain and daily pain. The treatment group was found to have lower levels of ankle stiffness than the placebo group (2.97 vs 3.58) although this difference was not statistically significant ($p=0.21$). Ankle stiffness was found to reduce with time. Although the relationship between time and stiffness was not significantly different between groups overall, the treatment group was found to have significantly lower levels of ankle stiffness on days six and seven ($p<0.05$).

LEG VOLUME

Leg volume was found to be normally distributed and as such was analyzed using a repeated measures analysis of variance adjusting for volume at baseline, age, gender and which leg (left or right). The average volume in the placebo group was found to be slightly higher than the treatment group (4691 vs 4633) although this difference was not statistically significant ($p=0.19$). No significant differences could be found between groups at specific time points. No variable (apart for baseline volume) was found to be statistically significant in predicting leg volume.

SERUM CREATININE KINASE

Serum Creatinie Kinase (CK) measures were found to follow a log-normal distribution. After a logarithmic transformation, the variable logCK was then analyzed using a repeated measures analysis of variance adjusting for logCK at baseline, age and gender. The geometric mean of the placebo group was found to be higher than that of the treatment group (256 vs 171) although the difference was not statistically significant ($p=0.21$). No significant differences could be found between groups at specific time points. Apart from baseline logCK, age was the only other variable found to have an effect on CK with an increase in age leading to a decrease in CK, although this did not reach statistical significance ($p=0.08$).

DISCUSSION

This study tested the efficacy of PEMF on experimentally induced delayed onset muscle soreness under double-blind placebo-controlled conditions. Results of the placebo treatments indicate that the methods used for inducing and assessing exercise-induced muscle injury were effective. Pain increased immediately after the exercise session, peaking at around day three and then declined until full remission at around day eight. Similarly ROM and mechanical pain threshold reduced immediately after exercise and began to recover after day three. This data is consistent with the time course of changes reported in the literature. The fact that no significant difference in serum CK was found between the groups suggests that equal injury was produced in both the placebo and control groups.

Results of comparisons between active and placebo treatments demonstrate that PEMF is effective in treating exercise induced muscle injury. The subjects who received active treatments experienced less pain and stiffness associated with DOMS as well as improvements in their range of motion and mechanical pain threshold. Most notable are the results on days four to seven.

It seems that the active PEMF treatment not only reduced the pain and stiffness associated with DOMS, but also reduced the overall recovery time with the treatment group appearing to reach full recovery at least one day earlier. It is interesting to note that in both groups a training effect was observed with regards to mechanical threshold and range of motion so that after one week, subjects had greater mobility and less tenderness than before commencing the trial.

The finding that the application of PEMFs is able to effectively treat exercise-induced muscle injury seems to raise more questions than it answers. From a clinical perspective PEMF therapy appears to be a relatively well tolerated, easy to administer, and cost effective therapy with no obvious adverse effects. However many questions remain to be answered, such as: What are the optimum treatment characteristics in terms of intensity and frequency of stimulation as well as the timing and duration of treatments? What are the conditions most suited to this type of treatment? Is this treatment method equally effective for other types of sports injuries, other soft tissue diseases or other painful conditions in general? Are there long-term effects of PEMF?

In addition to unanswered clinical questions, many fundamental questions are also raised by the success of this therapy. As yet it is unclear as to the mechanism of action of PEMF and whether these effects are mediated through the magnetic fields or induced electric fields? Furthermore it is uncertain whether PEMFs purely modulate pain perception or whether they play a more primary role in altering the inflammatory response, or effecting changes in membrane permeability, the structural or functional characteristics of proteins or by altering other, less well characterised, phenomenon? It would certainly be interesting to do further studies to characterise the cellular changes involved, as well as studies to determine the relationship between induced electric fields and the piezoelectric properties of collagen.

Results of this study suggest that Pulsed Electromagnetic Fields reduce muscle injury and pain and enhance recovery time when applied to muscles after eccentric exercise. These findings raise questions as to optimal treatment characteristics of these devices as well as their underlying biological effects.

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REFERENCES

SAS. 1999. Cary, NC, USA, SAS Institute Inc.

Aaron, R.K. and Ciombor, D.M. (1993) Therapeutic effects of electromagnetic fields in the stimulation of connective tissue repair. *J.Cell Biochem.* **52**, 42-46.

Armstrong, R.B. (1984) Mechanisms of exercise-induced delayed onset muscular soreness: a brief review. *Med.Sci.Sports Exerc.* **16**, 529-538.

Bassett, C.A. and Becker, R.O. (1962) *Science* **137**, 1063-1064.

Binder, A., Parr, G., Hazleman, B. and Fitton-Jackson, S. (1984) Pulsed electromagnetic field therapy of persistent rotator cuff tendinitis. A double-blind controlled assessment. *Lancet* **1**, 695-698.

Brown, S.J., Child, R.B., Day, S.H. and Donnelly, A.E. (1997) Indices of skeletal muscle damage and connective tissue breakdown following eccentric muscle contractions. *Eur.J.Appl.Physiol.* **75**, 369-374.

Craig, J.A., Barlas, P., Baxter, G.D., Walsh, D.M. and Allen, J.M. (1996a) Delayed-onset muscle soreness; lack of effect of combined phototherapy/low intensity laser therapy at low pulse repittion rates. *J Clin Laser Med Surg* **14**, 375-380.

Craig, J.A., Cunningham, M.B., Walsh, D.M., Baxter, G.D. and Allen, J.M. (1996b) Lack of effect of transcutaneous electrical nerve stimulation upon experimentally induced delayed onset muscle soreness in humans [see comments]. *Pain* **67**, 285-289.

Dudley, G.A., Czerkawski, J., Meinrod, A., Gillis, G., Baldwin, A. and Scarpone, M. (1997) Efficacy of naproxen sodium for exercise-induced dysfunction muscle injury and soreness. *Clin.J.Sport.Med.* **7**, 3-10.

Fischer, A.A. (1987) Pressure algometry over normal muscles. Standard values, validity and reproducibility of pressure threshold. *Pain* **30**, 115-126.

Fridén, J., Sfakianos, P.N. and Hargens, A.R. (1989) Blood indices of muscle injury associated with eccentric muscle contractions. *J.Orthop.Res.* **7**, 142-145.

Gleeson, M., Almey, J., Brooks, S., Cave, R., Lewis, A. and Griffiths, H. (1995) Haematological and acute-phase responses associated with delayed-onset muscle soreness in humans. *Eur.J.Appl.Physiol.* **71**, 137-142.

Hasson, S.M., Mundorf, R., Barnes, W., Williams, J.H. and Fujii, M. (1990) Effects of pulsed ultrasound versus placebo on muscle soreness perception and muscular performance. *Scan.J.Rehab.Med.* **22**, 199-205.

Ieran, M., Zaffuto, S., Bagnacani, M., Annovi, M., Moratti, A. and Cadossi, R. (1990) Effect of low frequency pulsing electromagnetic fields on skin ulcers of venous origin in humans: a double-blind study. *J.Orthop.Res.* **8**, 276-282.

Jensen, K. (1990) Quantification of tenderness by palpation and use of pressure algometers. In: Fricton, J.R. and Awad, E., (Eds.) *Advances in Pain research and Therapy*, 17 edn. pp. 165-181. New York: Raven Press, Ltd]

Jones, C., Allen, T., Talbot, J., Morgan, D.L. and Proske, U. (1997) Changes in the mechanical properties of human and amphibian muscle after eccentric exercise. *Eur.J.Appl.Physiol.* **76**, 21-31.

Lecomte, J.M., Lacroix, V.J. and Montgomery, D.L. (1998) A randomized controlled trial of the effect of naproxen on delayed onset muscle soreness and muscle strength. *Clin.J.Sport.Med.* **8**, 82-87.

MacIntyre, D.L., Reid, W.D. and McKenzie, D.C. (1995) Delayed muscle soreness. The inflammatory response to muscle injury and its clinical implications. *Sports Med.* **20**, 24-40.

Melzak, R. (1975) The McGill Pain Questionairre: major properties and scoring methods. *Pain* **1**, 277-299.

Newham, D.J., Jones, D.A. and Edwards, R.H. (1986) Plasma creatine kinase changes after eccentric and concentric contractions. *Muscle Nerve* **9**, 59-63.

Paddon, J.D. and Quigley, B.M. (1997) Effect of cryotherapy on muscle soreness and strength following eccentric exercise. *Int.J.Sports Med.* **18**, 588-593.

Price, D.D., McGrath, P.A., Rafii, A. and Buckingham, B. (1983) The validation of visual analogue scales as ratio scale measures for chronic and experimental pain. *Pain* **17**, 45-56.

Pyne, D.B. (1994) Exercise-induced muscle damage and inflammation: a review. *Aust.J.Sci.Med.Sport.* **26**, 49-58.

Sisken, B.F. and Walker, J. (1995) Therapeutic Aspects of Electromagnetic Fields for Soft-Tissue Healing. In: Blank, M., (Ed.) *Electromagnetic Fields: Biological Interactions and Mechanisms*, pp. 277-285. Washington DC: American Chemical Society]

Stiller, M.J., Pak, G.H., Shupack, J.L., Thaler, S., Kenny, C. and Jondreau, L. (1992) A portable pulsed electromagnetic field (PEMF) device to enhance healing of recalcitrant venous ulcers: a double- blind, placebo-controlled clinical trial. *Br.J.Dermatol.* **127**, 147-154.

Todd, D.J., Heylings, D.J., Allen, G.E. and McMillin, W.P. (1991) Treatment of chronic varicose ulcers with pulsed electromagnetic fields: a controlled pilot study. *Ir.Med.J.* **84**, 54-55.

Trock, D.H., Bollet, A.J., Dyer, R.H.J., Fielding, L.P., Miner, W.K. and Markoll, R. (1993) A double-blind trial of the clinical effects of pulsed electromagnetic fields in osteoarthritis [see comments]. *J.Rheumatol.* **20**, 456-460.

Vickers, A.J., Fisher, P., Smith, C., Wyllie, S.E. and Lewith, G.T. (1997) Homoeopathy for delayed onset muscle soreness; a randomised double-blind placebo controled trial. *Br J Sports Med* **31**, 304-307.

Wilson, D.H. (1972) Treatment of soft tissue injuries by pulsed electrical energy. *British Medical Journal* **2**, 269-270.