

The Use of a Specific Pulsed Electromagnetic Field (PEMF) in Treating Early Knee Osteoarthritis

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Introduction

Over the past four decades external physical fields have been used to stimulate bone formation in non unions and delayed fracture unions as well as to stimulate more rapid healing in the presence of a fracture where healing is anticipated¹. Despite multiple reports on clinical success there continues to be controversy as to the effectiveness of these fields². A recent review of the effect of electromagnetic fields on knee OA concluded that, although clinical scores improved, effects on pain were equivocal³.

Osteoarthritis of the knee is a leading cause of disability and loss of independence. Pharmacologic interventions used to manage OA pain have variable success and can produce considerable side effects. Non-pharmacological approaches include capacitively coupled electric fields which are used clinically in end stage knee OA and require the use of two electrodes in skin contact across the knee for 10 hours each day. The time required to notice a clinically significant difference may be as long as 75 days. Capacitively coupled fields appear to delay the time to total joint replacement in some individuals⁴.

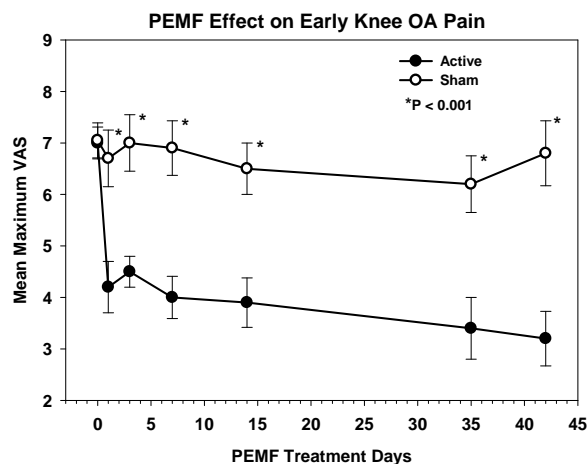
Inductively coupled pulsed electromagnetic fields (PEMF) provide a non-invasive, no-touch means to apply an electric field which can target intra and extracellular biochemical pathways. PEMF signals have recently been configured to modulate Ca²⁺ binding to calmodulin (CaM), followed by Ca/CaM binding to an enzyme such as constitutive nitric oxide synthase (cNOS) which leads to transient nitric oxide (NO) release⁵. Manipulation of this pathway is known to be anti-inflammatory⁶. Direct evidence that this PEMF signal can modulate this pathway has been reported for chondrocytes⁷, as well as neuronal cells in culture, and in a rat thermal injury model of cardiac ischemia⁸. Strong indirect clinical evidence has also been reported for post-surgical pain relief, chronic wound repair, and for cardiac myopathy patients with chronic angina⁹. This study examined whether the use of such a PEMF signal could prove effective in reducing pain in degenerative joint disease.

Methods

In a double-blind, randomized placebo-controlled study a total of 37 patients (19 active, 18 sham) started treatment. Selection required an initial max VAS score > 4, at least 2 hours of standing activity in a physical occupation, and no recent interventions such as cortisone injections or surgery. A PEMF signal, configured *a priori* to modulate Ca²⁺ binding to CaM, and consisting of a 7 msec burst of 6.8 MHz sinusoidal waves repeating at 1/sec with 0.05 G peak amplitude, in a portable battery operated device (Ivivi Technologies, Montvale, NJ) was used for 15 minutes twice daily, or as needed for pain relief. The device was light weight and patients could easily position the coil directly over the knee, even over clothing. Minimum and maximum VAS scores were obtained at baseline (day 0) and daily for the first 14 days and from day 29 to day 42. Results were analyzed using Mann-Whitney, ANOVA or repeated measures ANOVA, as appropriate. Significance was accepted at P ≤ 0.05. Data is displayed ± SEM.

Results

All patients continued PEMF treatment to day 14. Thereafter, 31 (16 active, 15 sham) at day 35, and 28 (16 active, 12 sham) at day 42, were available for analysis. The devices were well tolerated and no adverse events were reported. The results show PEMF caused a significant decrease in mean maximum VAS to approximately 45% of mean start VAS for the treated group by the end of day 1, which gradually fell to 55% of mean start VAS (P < 0.001). In contrast, there was no significant decrease in mean maximum VAS vs mean start VAS at any time point in the sham group (P = 0.555). There was no significant difference in mean start VAS between the active and sham groups (Active = 7 ± 0.31, Sham = 7.1 ± 0.34, P = .903). Results are summarized in the figure.



Discussion

In persons with knee OA, bone attrition, bone marrow lesions, synovitis/effusion, and meniscal tears are all causes of knee pain¹⁰. Bone marrow lesions (edema) manifest an inflammatory response to bone injury attributable to OA. It has been proposed that NO produced via the CaM-dependent cNOS pathway can relieve OA pain by increasing circulation, decreasing nerve irritation, and decreasing inflammation¹¹. The rapid onset response in the active group suggests a mechanism of action of PEMF that is anti-inflammatory, via the CaM/cNOS pathway which produces an initial rapid and transient release of NO leading to vaso and lymph dilatation⁸. This could cause a rapid reduction of bone marrow edema with the concomitant rapid reduction of pain observed here. The slow decay of pain thereafter in the active group may be related to a longer term PEMF effect on the progression or even resolution of the disease process via the known PEMF effects on growth factor release¹². Future studies will be required to assess this. The patient population treated did not have end stage disease. Involved individuals were actively employed and had to be on their feet at least two hours a day. PEMF treatment time is short (15 min) and did not interfere with work or off-work activities. Certainly the results are promising enough to warrant further larger studies which include two 6 week trial periods in each of which there is a random chance of active versus sham coil.

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