Table 1. *Numbers of patients with OA observed at each phase of the study*

	Numbers of Patients	
	Treated	Placebo
Entered into study	15	12
Evaluated at middle of treatment	14	11
Evaluated at end of treatment	14	11
Evaluated at one month followup	10	10

the active treatment and the placebo groups in any of the subjective patient variables or the physician examination variables evaluated.

Improvement occurred in each variable followed in the treated group, and data analyzed as matched pairs showed significant differences for the data for each variable at the midpoint of treatment, the end of treatment and one month after treatment. The patients who were followed showed continued improvement during the month after completion of treatment (Table 2A).

The placebo treated group showed some improvement from baseline in each variable, the change not reaching statistical significance for any of the observations (Table 2B).

The actively treated group averaged 34% improvement in the mean value for each variable evaluated at the midpoint of therapy, and 36% at the end of treatment; by one month after treatment ended, improvement averaged 47%; Figure 1A shows the percentage improvement in each of the 6 variables observed for the treated patients. Among the patients in the placebo group, improvement averaged 8% at the midpoint, 10% at the end of treatment, and 14% one month later (Figure 1B).

The overall assessment of improvement by the physician observer at the midpoint, end of treatment and one month after completion of treatment for treated and placebo groups is shown in Table 3.

The degree and frequency of improvement for upper extremity and lower extremity joints was similar.

No patient reported any increase in the use of their usual medications during the period of observation. Two patients, both in the treatment group, reported discontinuation of usual medications (ibuprofen, 800 tid in one, and pentazocine, 50 mg prn in the other); no placebo patient reported any change in medication.

Laboratory data at the end of treatment showed no changes in any tests including CBC, ESR, serum electrolytes, BUN, creatinine, and tests of liver status. No patient reported symptoms suggestive of toxicity nor was any toxicity observed by the physician evaluators.

The study was not designed for crossover analysis of an active treatment phase for the placebo treated patients; at the completion of observations, however, placebo patients were informed of the nature of their "treatment" and offered the opportunity to have active treatment in an unblinded fashion, and 7 did so. New baseline and followup observations were

Table 2. *Observations on actively treated and placebo-treated patients at each point. Figures are numbers of patients observed at each point (n) mean for groups*

Table 2A. Actively treated patients	Baseline	Midpoint	End of Treatment	1 mo Later
n =	15	14	14	10
Overall severity of pain (scored on 10 cm visual analog scale)				
Mean	7.65	4.28	3.80	3.55
SEM (±)	0.6405	0.88	0.82	1.08
p =		0.0215	0.0023	0.0052
Difficulty score with most troublesome ADL (scale: 1 to 5)				
Mean	4.27	3.25	3.07	2.80
SEM (±)	0.20	0.25	0.31	0.29
p =		0.0098	0.0020	0.0078
Pain with most troublesome ADL (scale: 1 to 5)				
Mean	3.90	2.89	3.14	2.40
SEM (±)	0.12	0.22	0.29	0.27
p =		0.002	0.0313	0.0078
Worst discomfort in previous week (scored on 10 cm visual analog scale)				
Mean	8.14	5.36	5.03	4.03
SEM (±)	0.58	0.74	0.83	0.94
p =		0.0046	0.0009	0.0059
Pain on joint motion by MD exam (scale: 1 to 5)				
Mean	3.47	2.07	2.29	1.85
SEM (±)	0.21	0.29	0.38	0.26
p =		0.002	0.0195	0.0117
Joint tenderness by MD exam (scale: 1 to 5)				
Mean	3.57	2.29	2.00	1.40
SEM (±)	0.33	0.29	0.29	0.22
p =		0.0034	0.0024	0.0039
Table 2B. Placebo-treated patients				
n =	12	11	11	10
Overall severity of pain (scored on 10 cm visual analog scale)				
Mean	8.07	7.23	7.32	7.10
SEM (±)	0.75	0.55	0.47	0.35
p =		> 0.3	> 0.6	> 0.5
Difficulty score with most troublesome ADL (scale: 1 to 5)				
Mean	3.88	3.77	3.59	3.65
SEM (±)	0.15	0.18	0.24	0.26
p =		> 0.6	> 0.4	> 0.6
Pain with most troublesome ADL (scale: 1 to 5)				
Mean	3.92	3.77	3.50	3.45
SEM (±)	0.15	0.23	0.20	0.26
p =		> 0.6	> 0.2	> 0.2
Worst discomfort in previous week (scored on 10 cm visual analog scale)				
Mean	8.18	7.16	6.65	6.82
SEM (±)	0.59	0.63	0.79	0.82
p =		> 0.4	> 0.3	> 0.3
Pain on joint motion by MD exam (scale: 1 to 5)				
Mean	3.25	2.86	2.82	2.60
SEM (±)	0.28	0.38	0.42	0.42
p =		> 0.3	> 0.4	> 0.2
Joint tenderness by MD exam (scale: 1 to 5)				
Mean	3.00	2.73	2.95	2.45
SEM (±)	0.44	0.36	0.32	0.26
p =		> 0.8	> 0.9	> 0.8

SEM = standard error of the mean, and p value is for change from baseline for that variable.

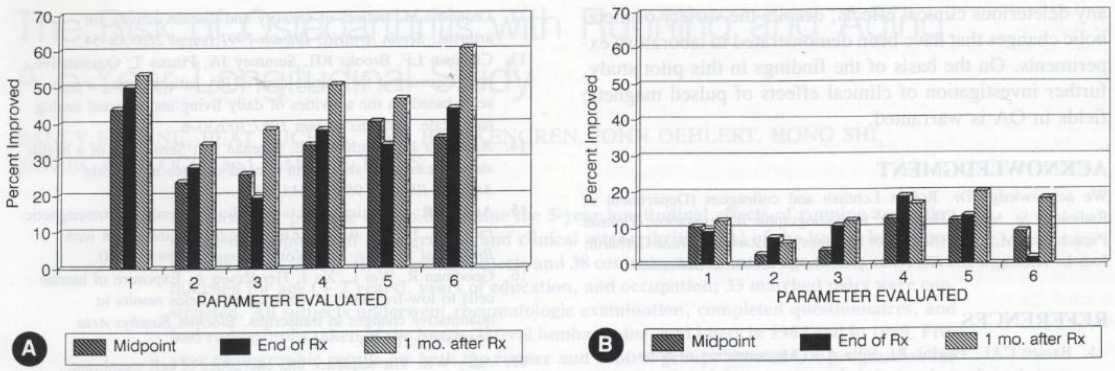

Fig. 1. Percent improvement, defined as difference between baseline value and value at each specific observation point, divided by baseline value (times 100). Numbered observations shown included (1) Overall severity of pain. (2) Difficulty performing ADL identified as the most troublesome by the patient before therapy was begun. (3) Pain generated by the most troublesome ADL. (4) The worst discomfort experienced in affected joint area in the past week. (5) Pain on motion of the treated joint detected by the examining physician. (6) Tenderness of study joint detected by the examining physician. 1A: Treated patients; 1B: Placebo patients.

Table 3. *Assessment of improvement by observing physician at midpoint of treatment, end of treatment and one month after completion of treatment. p Value is for difference between treated and placebo groups*

	Midpoint	End of Treatment	1 mo Later
Treated patients			
Mean	2.71	2.71	3.30
SEM (±)	0.27	0.37	0.45
Placebo patients			
Mean	1.73	1.86	1.75
SEM (±)	0.27	0.47	0.34
p =	0.0175	0.1611	0.0134

made on these patients. As a group, these patients showed improvement during the active treatment phase but, in view of the small numbers involved, statistically significant changes that occurred did not occur in all of the variables followed.

Radiographs were graded as to severity of the OA. There were too few cases in the treated group to permit meaningful statistical analysis of the response according to radiological criteria of severity. It is possible to say, however, that 5 patients with radiologic grade 3 and 4 disease obtained good or excellent responses according to physician assessment at the last observation, and thus that advanced disease does not preclude symptomatic benefit from this form of therapy.

DISCUSSION

The results of our prospective double blind study of PEMF treatment show beneficial effects in the amelioration of symptoms, subjective improvement in functional ability and decrease in objective findings in a small group of patients with OA. The benefit seemed to continue for at least the first month after completion of treatment. Furthermore, no toxicity was observed.

This application of PEMF therapy is not similar to other physical modalities of treatment, such as ultrasound, TENS, diathermy, moxibustion, etc. The PEMF generated by the device used in our study differs from the device used in the treatment of unhealed fractures in that it generates a lower frequency (<30 Hz vs 72 Hz), as well as differing in pulse and wave form characteristics. The extremely low frequency pulsed magnetic fields used in these studies, as well as those used in laboratory experiments, are too weak to work through a mechanism such as thermal effect, dielectric breakdown, particle displacement or electrophoresis. Mechanisms which have been suggested include some form of induced resonance of outer shell electrons, an effect on cell membrane receptors or on other endogenous processes, such as an effect on ion flux, but these suggested mechanisms lack experimental substantiation[15-18]. Evidence exists that pulsed magnetic fields can modulate the actions of hormones, antibodies, and neurotransmitters at surface receptor sites of a variety of cell types[15]. Effects on fibroblast, chondrocyte and osteocyte metabolism and lymphocyte functions have been reported. Augmentation of mRNA and protein synthesis has been reported in several tissue culture symptoms[16,17,19-25].

Since the factors responsible for the pain in patients with OA are varied and often uncertain in an individual patient, an attempt to delineate the mechanism of pain relief brought about by this form of therapy in relation to known biological effects of pulsed magnetic fields would be purely speculative.

This form of nonionizing radiation is not known to have